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HOLTH'S IRIDENCLEISIS ANTIGLAUCOMATOSA

FOLLOW-UP EXAMINATION OF ONE HUNDRED AND TWENTY-TWO EYES FROM SIX TO ONE HUNDRED AND FIFTY-NINE MONTHS AFTER OPERATION *

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In a paper read before the ophthalmologic congress in Vienna in August, 1921, I [1] gave an account of my first 30 iridencleises performed in private practice, as well as 27 cases of chronic glaucoma in which Elliot's trephining was done. All operations were performed in Drammen. The results of the iridencleises were investigated from twelve to seventy-five months after the operation. The effect of both methods seemed to be the same in regard to the relief from intra-ocular tension. Iridencleisis proved superior to Elliot's operation, however, in the preservation of vision and the field of vision. A demonstration of the operation before several members of the congress some days afterward at the Second University Ophthalmological Clinic led to the trial of the method there. Pillat gave an account of the results before the German Ophthalmological Congress in Heidelberg in 1927, and in a more detailed monograph in 1928.[2] In his first publication he recorded good results in 70 per cent of 80 eyes operated on for chronic glaucoma. In his monograph he said that he thought as many as 87 per cent of 100 patients were cured by the operation.

Several reasons, which I cannot discuss here, made me give up all forms of sclerectomy. But as I had been Holth's pupil from the time that he, as clinical chief of the ophthalmologic section of the Government Hospital (Rikshospitalet) in Oslo, was my teacher in the treatment for glaucoma, I tried his different methods of sclerectomy

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* Read in an abbreviated form before the Eighth Northern Ophthalmological Congress in Helsingfors on June 25, and before the Ophthalmological Congress in Oxford on July 9, 1931.

1. Gjessing, H. G. A.: Ueber die Iridencleisis antiglaucomatosa cum Iridotomia meridionali nach Holth, Verhandl. d. ausserord. Tag. d. ophth. Gesellsch., Wien (1921), 1922, p. 76.

2. Pillat, Arnold: Erfahrungen mit der Holth'schen Iridencleisis antiglaucomatosa, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **46**:70, 1927; Die Filtrationsnarben-bildenden Glaukomoperationen mit besonderer Berücksichtigung der Iridencleisis antiglaucomatosa Holth, Ztschr. f. Augenh., 1928 (supp.), p. 1.

with punch forceps. The results of the few sclerectomies of this kind that I performed did not encourage me to continue. However, I became a firm adherent of the first method that Holth had inaugurated, iridencleisis, which became my "method of choice" in operating for chronic glaucoma. In the few acute cases that have occurred in my private practice I have used total iridectomy. I have always made the incision in the sclera with the bent stop keratome. Without directly pulling the pillars of the iris into the wound, I have not given myself too much trouble to replace them lege artis. As a rule, the result has been good and permanent. None of these total iridectomies for acute glaucoma has been included in this record.

In 1923, before the Oxford Congress I reported 61 iridencleises. At the same time I briefly described the technic of the operation.[3] The patients were reexamined from six to one hundred and sixteen months after the iridencleisis. I thought myself justified in stating that I had obtained the following permanent results without using miotics: normal tension in 49 cases (about 80 per cent), preservation of the field of vision in 47 (77 per cent) and unaltered vision in 50 eyes (about 80 per cent).

I continued to use iridencleisis and kept in touch with my glaucomatous patients. In July, 1927, I reported before the congress in Oxford, on the results of iridencleisis shown at reexamination in 92 of 113 cases of chronic glaucoma; all the patients were operated on in private practice in Drammen. Most of the operations were performed with meridional iridotomy, as Holth[4] described in his first publications in 1906 and 1907. In only a few cases was total iridectomy done, the iris pillars being included in the wound.

I tried Holth's peripheral lobular iridencleisis in 8 cases, in most of which it gave good results. I employed it only in cases in which the field of vision was very limited and the tension high. I kept the sphincter intact in order to get a stronger effect from miotics. On 2 patients I performed only a very peripheral basal iridectomy without inclusion of the iris pillars, as von Hess recommended. The tension remained normal for four and seven months respectively. Then it increased again. For some time it could be normalized by means of miotics. Later it increased in spite of large and frequent doses of pilocarpine. One of the patients was operated on again (case 9, table 2); a total iridectomy was done with inclusion of both iris pillars in

3. Gjessing, H. G. A.: Clinical Examinations of Fistula or Filtering Scars After Some Operations, Especially After Holth's Iridencleisis Combined with Meridional Iridotomy, Tr. Ophth. Soc. U. Kingdom **43**:616, 1923; Resultater av iridenkleiseoperationer ad modum Holth, Norsk mag. f. lægevidensk. **88**:737, 1927.

4. Holth, S.: Iridencleisis cum Iridotomia Meridionali, Arch. Ophth. **4**:803 (Dec.) 1930.

the wound, as he had incipient cataract. The other patient disappeared from observation after eight months.

The results of my investigations in 1927 were: In 77 cases, or 83.7 per cent, tension was normalized without the use of miotics. I obtained the same figures for the vision. The field of vision was preserved in 77 cases, or 76 per cent.

From one point of view these findings—although apparently good—were discouraging: In a previous article [5] I had maintained that glaucoma was cured when iridencleisis had relieved the hypertension, and the visual field and vision were preserved. Two patients, however, who appeared to be cured when examined fifty and seventy-five months, respectively, after the operation did not continue to show good results. In one case the tension increased. The blind painful eye had to be removed one hundred and thirty-two months after the iridencleisis. The other patient still had normal pressure one hundred and two months after operation, but for no demonstrable reason, vision and the visual field were lost.

In one respect the investigations were encouraging: They seemed to show that cataract does not often follow iridencleisis, as Enroth [6] found is frequently the case after Elliot's trephining. Thomas Harrison Butler,[7] of Birmingham, stated in a letter that he had employed the method of Holth in a number of cases of chronic glaucoma since 1921. During the period from 1919 to 1929 he had obtained good results in 82 per cent of his cases. He had not seen any case of cataract develop after iridencleisis. Later I shall go into further details regarding his findings. The reason is unquestionably to be sought in the fact that following iridencleisis performed by Holth's technic, as against that of Weekers,[8] the tension never sinks to such a hypotonia as after trephining.

I must confess that I had a case of traumatic cataract following operation: A man had a very shallow anterior chamber. When the bent keratome was inserted into it, the iris was captured by its points and, undoubtedly, the anterior capsule of the lens was wounded. Though he left the infirmary with an apparently clear lens, the patient returned eight weeks afterward with a total cataract in that eye. Later, an extraction was performed through an incision in the clear cornea, after

5. Gjessing, H. G. A.: Further Investigations into the Results of Iridencleisis Operations by Holth's Method, Particularly as Regards the Method Combined with Meridional Iridotomy, Acta ophth. **5**:149, 1927.

6. Enroth, Emil: Ueber Katarakt nach Elliots Operation, Acta ophth. **5**:113, 1927.

7. Butler, T. H.: Personal communication to the author on March 31, 1931.

8. Weekers, L., and Hubin, R.: Résultats obtenus par l'enclavement de l'iris dans le glaucome, Arch. d'opht. **48**:186, 1931.

preliminary suturing of the cornea according to Liégard's technic. On reexamination, twenty-one months after the extraction, vision and the visual field were the same as before the first operation. The tension was normalized without miotics, viz., to 19 mm. of mercury.

It was, however, the two cases leading to amaurosis that made me ask myself: Does iridencleisis really assure a good result? Is not its apparent effect only a transient delay in the inevitable progress of the glaucoma?

My investigations were continued. The records of St. Josephs Hospital show that from August, 1911, to Feb. 25, 1931, I performed 250 operations on 231 patients with glaucoma. Of these operations, 152 were iridencleises; others included: 32 Elliot operations, 7 iridotases by Borthen's method, 33 iridectomies of various kinds—with or without cutting of the sphincter—and a few sclerectomies. In some cases of absolute glaucoma, enucleation was performed.

All the iridencleises were performed on eyes with chronic glaucoma. With these were included not only the comparatively few cases of glaucoma simplex, but also the more numerous ones in which there had been traces of injection at intervals. There was also a case of subacute glaucoma: A woman, aged 47, had had periods of blurred vision for several years. Suddenly a typical acute attack developed, which was checked with pilocarpine. Total iridectomy was performed, the pillars of the iris being included in the wound. The result was excellent: Examination sixty-nine months after operation showed improved vision—6/6 as against 6/10 before the operation—and an unaltered visual field. Without miotics the tension was 19 mm. of mercury. I used Holth's operation in a case of buphthalmos.

I have never performed iridencleisis twice on the same eye, but in three cases it served as a method of reoperation, after other procedures had failed. In one of these cases I myself had performed an Elliot trephining without effect. In another, as mentioned, I had tried a basal iridectomy without effect. Through a subconjunctival keratome incision I drew one iris pillar into the wound. There was a transient edema over the included iris pillar and depression of the tension. Soon, however, the latter increased again in spite of the energetic use of pilocarpine. The patient, a woman, aged 64, refused to undergo a third operation. The eye became amaurotic.

In my iridencleises since 1925, I have made a retrobulbar injection of 2 cc. of a 2 to 4 per cent solution of procaine hydrochloride, as recommended by Duverger;[9] in injected eyes I use a 4 per cent solution. To this I add 1 or 2 drops of epinephrine. In a visit to Glasgow, in

9. Duverger, C.: L'anesthésie locale en ophtalmologie, Paris, Masson & Cie, 1920, p. 59, fig. 7.

1927, I learned that the anesthetic effect could be increased by adding 1 per cent of antipyrine. I have used van Lint's [10] method of akinesia since 1920. However, during the last few years I have applied the preauricular modification recommended by Colonel Wright.[11]

Duverger's method of retrobulbar injection is of the greatest importance in every operation for glaucoma. Apart from ideal anesthesia, the injection produces a considerable decrease in tension when made twenty minutes before the opening of the anterior chamber. This decrease of intra-ocular pressure will combat the comparatively frequent retinal hemorrhages attending all operations for glaucoma and iridencleisis. In several cases I saw these hemorrhages in the fundus—especially near the papilla and macula—when the patient left the hospital, usually from eight to ten days after the operation. Gunnufsen [12] and Uhthoff [13] made similar observations. As a rule, the hemorrhages are small and insignificant, but this is not always the case: An iridencleisis was performed on a patient whose other eye was blind (K. N., table 1, case 18). Before operation, vision was 6/8, but the pressure remained high (7.5/1) despite large doses of miotics. The visual field was normal. As a result of the operation there was considerable bleeding in the macular region, which greatly decreased vision. The hemorrhage resorbed, so that six months afterward vision had increased to 6/6. Another case was the most tragic of all those in which I performed operation. For a long period I had treated a patient with pilocarpine with good results; his other eye was blind (N. K., 78 years of age, table 1, case 17). As the field shrank, however, I had to operate on him. When he lay down on the operating table he had full vision of 6/8. During the iridencleisis a preretinal hemorrhage occurred, and the patient left the hospital completely blind. The tension then, as well as twenty-one months afterward, was 5.5/5 without miotics. Since I began to use the retrobulbar injection according to Duverger's method, I have not noticed retinal bleeding.

I have also followed the technic indicated by Holth, though I seldom suture the small conjunctival wound made by the 6 mm. keratome. In my opinion—and Holth agrees with me—it is unnecessary to insert stitches unless the Graefe knife is used. I shall go into further details about this question later. Since 1925, I have also used a cautious

10. van Lint: Paralysie temporaire provoquée dans l'opération de la cataracte, Ann. d'ocul. **151**:420, 1914.

11. Wright, R. H.: Administration and Professional Report of the Government Ophthalmological Hospital, Madras, for the Year 1928, printed 1929, p. 9.

12. Gunnufsen, T.: Ueber die operative Behandlung des primären Glaucoms, Klin. Monatsbl. f. Augenh. **57**:283, 1916.

13. Uhthoff, W.: Ueber Glaucomoperationen, Verhandl. d. ausserord. Tag. d. ophth. Gesellsch., Wien (1921), 1922, p. 6.

massage through the closed lids from the third to the fifth day after operation in order to obtain a slight disruption of the wound and looser scar tissue. After the patient leaves the hospital, he uses a 2 per cent solution of pilocarpine once or twice a day. He is ordered to return for observation after six months and not to use the drops on the day that he comes to be examined. If there is reason to believe that the tension will be normalized, he is ordered not to use miotics two days before observation. If the tension is normal and examination shows nothing unusual, the pilocarpine is discontinued, and the patient is ordered to come back to be examined every six months as long as he lives. Thus I have managed to keep under observation most of my patients on whom iridencleisis has been performed. I am sorry that I have not been able to do so with my glaucomatous patients who were not operated on, many of whom disappeared from observation after a time. The fact that my practice is within a rather limited territory, with easy communication, has facilitated my contact with patients on whom I have operated for glaucoma.

Thus I can report on 122 of my 155 iridencleises, performed in Drammen. I have not counted those done during the six months before this writing. The shortest time of observation following operation was six months; the longest, one hundred and fifty-nine months. All reexaminations were made by me personally. I have never taken into account letters from the patient's relatives or even from the regular physician. I have learned by experience that only a specialist can judge the results of an operation for glaucoma because of the many different factors involved.

First, the age of the patient plays a great part. An eye without any arteriosclerotic changes will give a better prognosis as to the retinal and visual condition than the eye of a man in his late seventies. However, typical juvenile glaucoma as a rule gives a worse prognosis than typical senile glaucoma. I must emphasize that even advanced age is not an absolute contraindication to an operation for high tension. Still one should be more conservative in dealing with an aged person, especially when there is vision in only one eye, and try as long as possible to see what miotics can do.

The condition of the field of vision plays an important part. If it is kept unaltered by the use of miotics, I do not operate even if the tension is not too much increased—especially in old persons. If the field shrinks, however, even with apparently normal tension, I do an iridencleisis as soon as possible. I shall return later to the subject of the border value of the tension. Of my 122 reexamined patients on whom iridencleisis was done, 10 showed a shrunken field of vision, in spite of normal tension without miotics. However, 7 of these

patients had a very limited field before. Only 3 had practically a normal field before operation (tables 1 and 3).

The duration of the disease also plays an important part. As a rule, the longer it is, the more the optic nerve will be damaged. Few patients observe the first symptoms, owing especially to absence of pain. Transient rainbow rings around the lights are easily overlooked by less intelligent persons. Thus, many of my patients come for examination only when the field of vision of their poorer eye is greatly shrunken. Statistics from private practice, therefore, will show more severe cases of glaucoma, because the patients are referred to the specialist by general practitioners, and do not often come directly, as to a public ophthalmologic clinic. As few of my patients with glaucoma have been able to tell me exactly when their complaint began, I have deemed it inadvisable to include this information in my tables. Regarding the sex of the reexamined patients, 41 were women and 76 men. This is probably without significance.

In the tables, tension I indicates an intra-ocular pressure the limit of which ranges between 3+ and 1, using the lowest weight (5.5) with the tonometer of Schiötz. In tension II there is a turn with weight 7.5, and in tension III with weights 10 or 15 as needed. The fields of vision are classified as follows: Field I is a normal field or one in which there is a shrinkage of not more than 20 degrees from the periphery in any one of the eight chief meridians. Field II has a defect which reaches the 20 degree circle in one or more of the eight chief meridians, while field III shows a shrinkage inside the 20 degree circle and includes the "spyglass" field. All the fields were taken with the grayish-white 2.5 mm. bead object of Holth's chord perimeter. The color limits were examined with his 10 mm. round celluloid flakes. All examinations were made in daylight on Förster's perimeter (radius, 31 cm.), placed about 1.5 meters before a large window. The visual acuity is classed as follows: Vision I is from 6/5 to 6/12, full; vision II is less than 6/12 and more than 6/60 and vision III is 6/60 or less.

CASES SHOWING NO RESULT

In 8 (32 per cent) of the 26 cases in which iridencleisis produced no result, a total iridectomy was performed. One or both iris pillars were drawn into the wound. I am sorry that I have not always noted this fact in my cases. The remaining 68 per cent of the patients underwent meridional iridotomy. However, these figures should be considered in relation to all the iridencleises performed. Of the 122 iridencleises followed by reexamination, only 20 (16 per cent) were performed with total iridectomy. This fact alters the point of view. About

TABLE 1.—*Observations on Cases Found Worse at Reexamination*

Case	Patient	Age at Operation	Eye	Operation	Reexamination	Before Operation			After Operation				Method	Comment
						Vision	Field of Vision	Tension	Vision	Field of Vision	Tension	Months		
1	B. J.	70	Left	3/ 6/16	2/ 7/25	6/ 8	I	5.5/1	0	0	15/4	132	Iridectomy	Good result for 50 mo.; then the tension increased
2	N. P.	63	Right	7/17/28	7/30/30	6/10	I	7.5/2	6/21	II	5.5/2	36	Iridotomy	The eye was excised
3	O. K.	54	Right	5/ 5/15	6/26/16	6/ 8	I	7.5/1	Less than 6/10	II	5.5/2	13	Iridectomy	Incipient cataract
4	B. J.	73	Right	2/19/17	3/ 2/26	6/ 6	I	5.5/1	6/36	II	5.5/2	109	Iridotomy	
5	T. M.	73	Right	2/ 7/17	6/ 3/24	6/10	II	10/3	Fingers at 2 meters	III	5.5/3+	89	Iridectomy	Incipient cataract
6	K. J.	54	Left	4/22/22	2/ 6/31	6/10	I	10/2	0	0	5.5/2	106	Iridectomy	Result partially worse, due to toxic amblyopia
7	E. O.	68	Left	12/18/28	2/ 4/31	6/18	III	5.5/1	4/60	III*	5.5/4	25	Iridectomy	
8	B. J.	68	Left	10/28/29	10/18/30	6/10	III	7.5/1	6/20	III*	5.5/5	12	Iridotomy	
9	F. M.	67	Right	8/ 3/23	2/19/26	6/10	I	5.5/2	6/18	III	5.5/3	30	Iridectomy	
10	T. J.	73	Left	5/ 5/19	6/ 4/25	6/12	II	7.5/2	6/36	III	5.5/2	73	Iridotomy	Tension very high without pilocarpine
11	G. G.	59	Right	6/24/29	1/30/31	6/18	I	5.5/1	Less than 6/36	III	5.5/4	19	Iridotomy	
12	G. H.	74	Left	5/ 3/22	12/ 3/28	6/ 6	III	5.5/1	6/60	III*	5.5/1	82	Iridotomy	
13	G. K.	71	Right	10/ 3/23	10/10/30	6/ 6	I	7.5/1	Light sense	0	5.5/1	84	Iridotomy	Did not come under control during the whole period of observation
14	J. J.	58	Left	3/15/21	4/21/23	6/15	II	10/3+	0	0	15/1	25	Iridectomy	Not under control during whole period of observation
15	K. P.	72	Right	1/ 6/21	12/14/30	6/ 8	I	5.5/2	Light sense	0	7.5/2	107	Iridectomy	Good result for 36 mo.
16	K. R.	8	Left	8/27/30	2/ 2/31	0	0	15/2	0	0	5.5/4	6	Iridotomy	Buphthalmos
17	N. K.	78	Left	9/24/24	10/20/27	6/ 8	III	7.5/1	0	0	5.5/5	21	Iridotomy	Bleeding in the macula
18	K. N.	68	Left	3/30/23	8/ 2/28	6/ 8	I	7.5/1	6/15	III	5.5/2	65	Iridotomy	
19	N. K.	63	Left	8/26/23	1/20/25	6/ 8	III	7.5/1.5	0	0	5.5/2	17	Iridotomy	Intra-ocular bleeding 5 days after operation
20	L. J.	74	Left	2/23/21	2/20/28	6/ 8	I	7.5/2	Less than 6/24	II	5.5/2	84	Iridotomy	
21	L. A.	78	Right	3/12/22	12/20/25	6/ 6	I	10/2	6/36	III	5.5/2	42	Iridotomy	
22	L. M.	75	Right	7/10/25	5/22/29	6/ 6	I	5.5/1	6/15	III	5.5/1	46	Iridotomy	Used pilocarpine during the whole time
23	N. O.	65	Left	5/ 5/29	5/ 2/30	6/15	I	5.5/1	6/24	III	5.5/4	12	Iridotomy	
24	R. S.	63	Right	1/21/20	5/14/30	6/10	I	5.5/1	6/20	I	5.5/7	125	Iridotomy	No reason could be found for decrease of vision
25	.....	..	Left	10/26/23	5/14/30	6/18	II	7.5/2	Less than	III	5.5/5.5	89	Iridotomy	No reason could be found for decrease of vision
26	O. O.	75	Left	5/ 6/30	2/ 1/31	6/ 8	I	5.5/1	6/18	I	5.5/3	8	Iridotomy	A great scotoma of Bjerrum had developed, just at the point of fixation

* Still more reduced.

85 per cent of all iridencleises, then, in which meridional iridotomy is performed result in apparent cure. Thus the figures for iridectomy are much more unfavorable than for iridotomy. My figures are too small for me to draw any stringent conclusions, but they point in a definite direction. Because of that fact Holth also kept this operation as his "method of choice." If one regards iridotomy anatomically, it is reasonable to assume that this operation gives better results regarding subconjunctival filtration. None of the fistula-forming material is lost. In slides from eyes operated on even six years before, Holth demonstrated a direct fistula from the anterior chamber to the subconjunctival tissue. The fistula was covered with uveal epithelium.

Concerning the iridencleises designated in table 1 as failures, the following facts are obvious: In 9 cases both vision and fields were unaltered and the tension was normal without miotics for a long time. Then and not till then, a decrease in vision started with or without an assignable reason. In all 9 cases the operation had at least a dilatory effect on the final catastrophal event, and for most of the patients this interval of good vision was not short. It lasted for eighty months in case 20, for seventy-two months in case 3 and for seventy months in case 1. In cases 4 and 15 the interval was thirty-six months. During this time the patients did not need miotics. One of them could keep the intra-ocular tension normal with pilocarpine during his whole life. In case 20 the tension was normal and the field of vision decreased for a long interval; the patient was then more than 80 years of age. The shortest interval of improved vision—only twelve months—occurred in case 9.

Only 7 of all my patients were less than 50 years of age; 21 were between 51 and 60. The age period from 61 to 70 included 48 persons; 43 were between 71 and 80 years of age and 3 were more than 80 years of age when they were operated on. How important a period of improved vision is to persons of such advanced age!

Opinion differs as to the interval that should elapse between operation and reexamination before one may speak of a successful operation for glaucoma. Even if there are some exceptions in my material, I think that one may consider a glaucomatous eye as saved when the tension is normal and the visual field and vision are unaltered, or almost so, thirty-six months following operation; at least I do not think it correct to set a shorter limit for persons below 70 years of age.

Retrobulbar anesthesia was used in cases 17 and 18. In case 19, bleeding occurred in the macular region the fifth night after operation. This, however, resorbed; six months later, vision was normal. The tension also was normalized without pilocarpine. Seventeen months after operation, however, although the pressure had increased but little,

vision had diminished to amaurosis. No reason for this catastrophe could be found. Before operation, however, the field of vision was very limited.

In the operations regarded as failures, one feature must be taken into account: The patients were all between 70 and 80 years of age. Even though I did not notice senile macular alterations, they may have been present, although insignificant. Neither can one eliminate the possibility of the presence of senile optic atrophy, described by Fuchs, which is not so rare as many ophthalmologists seem to think. This causes only a moderate loss of vision.

In only 3 cases did iridencleisis produce directly an unfavorable effect. Case 5 showed the rapid development of a preexistent cataract. As mentioned before, the patient in case 17 became amaurotic on the operating table. Case 18 was cited in my records for 1925 as showing a very good result. Only in one case (no. 14) did iridencleisis fail to produce a lowering of intra-ocular pressure. In all the other cases miotics had a better effect than before the operation.

TENSION

In my investigation on tonometry [14] based on measurements of the tension of 2,186 eyes in 1,095 persons, aged from 10 to 97 years—none of whom had any pain attributable to alteration of the intra-ocular pressure and most of whom were normal—I found that the lowest limit of the normal tension must be quoted as from 7 to 8, with weight 5.5. In certain persons—23 of whom were more than 50 years of age—I found a turn of the tonometric index above 6. But the upper limit must remain as Schiötz estimated it: 5.5/3. In this respect my findings are congruent with those of other investigators, especially Bernhard Cridland.

In two respects, Holth was right when he emphasized the use of the 6 mm. bent stop keratome as superior to the narrow knife in making the incision. With this keratome it is much easier to avoid the forming of subconjunctival scar tissue, which will prevent filtration. Holth demonstrated this fact anatomically. The scar tissue will develop much more easily when one makes a flap with the scissors like that in Elliot's trephining. Moreover, the keratome incision gives a much better superior scleral beak to cover the included iris and a better inferior scleral beak to cover the ciliary body than an incision made with the Graefe knife. 1. A good superior scleral beak will, to a high degree, prevent a late infection, while a thin conjunctival bleb at the limbus seldom occurs, provided that the operation is performed lege artis. The included iris

14. Gjessing, H. G. A.: Ueber Tonometrie, Festschr. f. Ernst Fuchs, Arch. f. Ophth. **105**:220, 1921; Tonometry, Brit. J. Ophth. **6**:452, 1922.

is covered with comparatively thick, well vascularized conjunctiva. 2. Both scleral beaks will also prevent the tension from being lowered so suddenly and to such a degree as in the Elliot trephining. This I think is of the greatest importance: The eye gradually adjusts itself to a moderate decrease of intra-ocular pressure within normal limits. This decrease does not always come directly after the operation, but often takes place during the following weeks or months. Holth himself stated that this happens in about 35 per cent of all cases after iridencleisis. Thus the patient leaves the hospital with an increase of pressure. However, in most of these cases the tension is lower than before, and the patient gives a stronger reaction to miotics.

Of my 122 cases of iridencleisis at the last reexamination, 9 showed a pressure of little lower than that usually considered as the normal limit. In 7 it was 5.5/7; in 1, 5.5/8, and in 1, 5.5/9. The patient in the last-mentioned case examined eight months after the operation, had the lowest tension that I have ever measured in persons who have undergone iridencleisis combined with meridional iridotomy.

I never saw cataract develop in an eye operated on by that method when the lens was clear before the operation. I except the case already mentioned, in which I fear I produced a traumatic cataract during the operation. The cause appears to be this gradual depression of the increased tension which never progresses to hypotonia, as often occurs in eyes operated on by Elliot's method. Neither Holth nor Harrison Butler has noticed it in their iridencleisis material. Butler, however, saw cataract develop in 10 per cent, and Enroth in about 29 per cent (26 of 29 eyes) of cases in which Elliot's trephining was done.

A drawback in using the bent stop keratome in making the subconjunctival tunnel is that a good trained assistant is required to hold the blunt double conjunctival hook. This trouble is avoided in the other Norwegian operation for glaucoma, the iridotasis of Borthen.[15] As I myself have performed only 7 of these operations—several, however, with good lasting results and observed over a very long period—I do not think it fair to this method, to that of Elliot or to Holth's aforementioned methods of sclerectomy to make any comparison with iridencleisis. It is impossible to draw any conclusion regarding the effectiveness of an operation for glaucoma from only a small number of cases. Thus I think that the results published by Galetski-Olin[16] concerning iridencleisis and iridotasis must be accepted with reservations, as her numbers are too small. Both the operations have the

15. Borthen, Johan: Operativ glaukombehandling: Iridotasis antiglaucomatosa, Norsk mag. f. lægevidensk., 1909, p. 704; Iridotasis antiglaucomatosa, Arch. f. Augenh. **68**:14, 1910.

16. Galetski-Olin, Hanna: Erfahrungen über Iridencleisis, Iridotasis, Sclerectomie und Trepanation, Klin. Monatsbl. f. Augenh. **68**:139, 1922.

same difficulty, viz., that an inexperienced operator will easily draw too much iris tissue into the wound. Holth tried to avoid this by constructing iris forceps with a regulating screw. His elbowed fixation forceps is also recommended for making the subconjunctival tunnel, because it enables the operator to make it longer. If too much iris tissue falls subconjunctivally, one is forced to excise a little of it after the iridotomy.

I am sorry to say that I have not noted the exact appearance of the scar except in my first 40 iridencleises. As mentioned before, after an iridencleisis performed lege artis one never sees a thin conjunctival bleb just at the limbus, as is regularly found after trephining. Contrary to the result in Elliot's operation, the phenomenon of Seidel usually is negative in Holth's operation. In only 3 between 50 and 100 patients examined with the fluorescein test did I find free fistulation. In all of them the incision was made too near the limbus and a thin-walled bleb had formed. As I pointed out in my paper in *Hygiea,*[17] such a bleb is not necessary to the maintenance of normal tension. It is not possible to draw conclusions as to the effectiveness of the operation from the appearance of the scar: The tension may be above normal in the presence of diffuse edema or even a bleb, as after the Elliot operation. A scar barrier may interfere with filtration. On the other hand, with no edema present, even after massage, iridencleisis often gives a successful result. Often one sees only two small black points extending subconjunctivally into the wound—the included iris pillars. In other cases, a good deal of iris tissue may be seen. The area surrounding the scar is then often diffusely pigmented. Whether this emigration of pigment from the uveal pigment is of any importance in the result and permits one to draw any conclusion regarding good filtration I do not venture to say. In my limited experience, however, those scars are best which give a diffuse edema that is increased a little by massage without forming a cushion.

I have already mentioned that the results of meridional iridotomy in iridencleisis will be somewhat different from those of total iridectomy with inclusion of one or both iris pillars in the wound. As none of the fistula-forming material is lost, the former method will give a greater tension-depressing effect. Also the cosmetic result is better, as the pupil is but slightly dislocated, and the patient thus avoids a disagreeable dazzling. This is seldom seen, even after a broad iridectomy, but now and then it may be troublesome.

Likewise, every oculist who consistently operates in each case of glaucoma as soon as it comes within his scope will obtain a better result than he who, like myself, operates only when miotics have proved

17. Gjessing, H. G. A.: Undersøgelser over Seidels fenomen, Hygiea, 1923, p. 230.

TABLE 2.—*Observations on Thirteen Considerably Improved Cases*

Case	Patient	Age at Operation	Eye	Operation	Reexamination	Before Operation			After Operation				Method	Comment
						Vision	Field of Vision	Tension	Vision	Field of Vision	Tension	Months		
1	F. O.	76	Left	4/16/28	5/10/29	2/60	III	7.5/1	6/60	III	5.5/1	11	Iridotomy	Field of vision remarkably enlarged
2	A. J.	49	Right	2/ 4/27	2/ 2/30	Fingers at 2 meters	III	10/2	6/24	III	5.5/6	36	Iridotomy	
3	F. A.	68	Left	1/16/28	7/21/28	Fingers at 3 meters	II	5.5/1	6/36	II	5.5/7	7	Iridectomy	
4	H. L.	63	Left	7/16/24	8/ 1/30	6/24	II	7.5/1	6/18	II	5.5/4	72	Iridotomy	Field of vision remarkably enlarged
5	M. H.	56	Left	1/30/26	7/30/30	6/20	III	7.5/1	6/10	III	5.5/6	49	Iridotomy	Field of vision remarkably enlarged
6	K. M.	69	Right	2/ 2/28	9/15/29	6/20	II	5.5/2	6/10	I	5.5/3	14	Iridotomy	Field of vision remarkably enlarged
7	R. P.	66	Left	5/14/29	1/12/31	Fingers at 2 meters	I	5.5/2	6/36	I	5.5/3.5	19	Iridotomy	Improvement due chiefly to optical effect of the iridotomy, a central lens opacity being avoided
8	M. R.	75	Left	4/12/27	5/ 7/28	6/15	I	7.5/1	6/10	I	5.5/7	11	Iridotomy	
9	A. L.	78	Right	10/ 6/16	8/14/17	Fingers at 2 meters	I	7.5/1	6/36	I	5.5/6	8	Iridectomy	Basal iridectomy, without iris inclusion, performed 5 mo. before
10	A. A.	74	Right	1/ 7/18	8/ 7/18	Less than 6/12	I	5.5/1	6/ 8	I	5.5/4	7	Iridotomy	
11	F. B.	71	Right	8/20/18	3/13/22	Less than 6/8	I	5.5/1	6/ 6	I	5.5/4	43	Iridotomy	
12	P. P.	48	Left	5/19/21	9/12/24	Less than 6/12	I	5.5/1	6/ 6	I	5.5/5	39	Iridotomy	
13	K. A.	66	Right	3/ 3/17	6/ 3/25	Less than 6/18	I	5.5/1	6/12	I	5.5/3+	99	Iridotomy	
Supplementary:														
	N. A.	73	Left	3/10/22	10/10/27	0	0	10/2	0	0	15/2	65	Iridotomy; cyclodialysis	Blind and painful eye at the first operation, which relieved the pain but not the pressure; Heine's operation brought the tension down to 7.5/2

useless. My records have shown that direct damage may appear after the operation. Therefore I must confess that I am possibly a little too conservative, especially when one eye is blind. If vision and the visual field can be maintained by drops, I delay operation as long as possible. However, there is a cogent reason for immediate operation, e. g., if the patient lives a great distance away or if the tension is not normalized even by large doses of miotics, I operate instantly.

CASES SHOWING IMPROVEMENT

All 13 cases quoted in table 2 showed considerable improvement. Unlike some other records, my tables are to be understood literally: Unaltered means that the patient showed the same field of vision as before the operation or that he read the same line on the Snellen chart as before; at least his vision must be decreased for more than one or two characters. Such a small decrease may be due to postoperative astigmatism, though this is usually small. If the patient reads a whole line less than before, the situation is regarded as worse. All the improved patients read at least one whole line more on Snellen's chart or Hertel's roller than before.

Except in 2 cases (nos. 1 and 13), the improvement occurred in all three components: tension, vision and visual field.

In case 1, eleven months after the operation the tension had increased to 5.5/1 in spite of pilocarpine given three times a day. Before iridencleisis the tension could not be lowered to less than 7.5/2 despite great doses of miotics. At reexamination, however, the field of vision was found to be enlarged nasally. At the same time, vision was increased. In case 13, ninety-nine months after operation the tension was 5.5/3 +; pilocarpine was therefore instilled twice a day; otherwise, considerable improvement was noted. Cases 4, 5, 6 and 12 all showed considerable improvement of the field of vision in all the chief meridians. However, in case 12 the field was good before operation. The improvement of vision in case 3 was perhaps due more to the subsequent extraction of a cataract than to iridencleisis alone, but after the latter the tension fell from 5.5/1 to 5.5/7.

Three of the patients cited in table 2 underwent total iridectomy and iris inclusion; the others, meridional iridotomy.

In the supplementary case, N. A., a man, aged 73, was blind before the operation. However, the tension could not be brought lower than to 10/2 (60 mm. of mercury), even with frequent doses of pilocarpine. Iridencleisis was performed. When he returned sixty-five months later, intra-ocular pressure had increased to 15/2 (about 100 mm. of mercury). He suffered great pain. Excision of the eye was suggested, but tentatively a cyclodialysis was performed. This brought the pressure down to 7.5/2 (about 45 mm. of mercury) and gave relief from pain. The

patient, now about 78 years of age, was given pilocarpine and sent home. He was ordered to come back if the pain returned, and have the eye enucleated.

Case 2 is one of the few in my material which is to be classified as juvenile glaucoma. Before iridencleisis the tension was 10/2 (60 mm. of mercury). A month before, the patient had had a typical attack of acute glaucoma. The intra-ocular pressure was then 15/2. Even a 2 per cent solution of pilocarpine, six times daily, did not diminish it to less than 10/2. At the last examination, thirty-six months after iridencleisis, the tension was 5.5/4 (about 19 mm. of mercury). She had then used pilocarpine twice a day for six months, after which the drops were discontinued. In spite of repeated requests to come for reexamination, she did not return. I have heard that she later developed diabetes.

ANALYSIS OF RESULTS

Analysis of the cases listed in table 3 as to tension, vision and field of vision on reexamination shows: Ninety-seven (79.5 per cent) patients had normal tension without miotics; 10 (8.2 per cent) had normal tension with miotics, and 15 (12.3 per cent) had increased tension in spite of miotics. Thus, in 87.7 per cent of cases tension was normalized by iridencleisis. Eighty (65.6 per cent) had unaltered vision; 17 (13.9 per cent) had increased, and 25 (21.2 per cent) had decreased vision. Thus in 79.5 per cent of the 122 patients vision was unaltered or even increased by Holth's operation. In 91 (76.6 per cent) the field of vision was unaltered; in 8 (6.6 per cent) it was increased, and in 23 (18.8 per cent) it was decreased. Thus, the field of vision was preserved or even increased in 83.2 per cent.

Computing the average of the results for the three factors, tension, vision and field of vision—possibly a justifiable objection may be made to this procedure when such incommensurable values are involved—I obtained a favorable result from iridencleisis in 83.5 per cent of cases.[18]

18. As already mentioned, Harrison Butler performed iridencleisis in a fair number of cases of chronic glaucoma. He obtained good results in 82 per cent of the cases, a figure very near those of mine and of Holth, who obtained 85 per cent in his first 130 iridencleises. Butler reported no late infection or excision. Noticeable also is the fact that Butler operated in 10 cases of acute glaucoma with iridencleisis with excellent results. Holth (footnote 4), during the last fourteen years, performed this operation in 6 cases of acute glaucoma, which is rare in Norway, also with admirable effect. I have pointed out (Acta ophth. **5**:152, 1925) that I believe the favorable lasting result of iridectomy in acute glaucoma is due to an involuntary inclusion of iris. The old Saxonian ophthalmologist Coccius also decided that inclusion of iris was the successful operative treatment for glaucoma. David Priestley Smith (Birmingham) wrote to me that he had used the operation of Holth since 1921. He was satisfied with it, especially because it never produced dangerous hypotonia, which he had often seen following Elliot's trephining.

TABLE 3.—*Observations on One Hundred and Twenty-Two Cases Before and After Iridencleisis by Holth's Method*

Before Operation										After Operation									
	Tension			Vision			Field of Vision			Reexamination, Number Months After Operation	Tension			Vision			Field of Vision		
											Normal								
No. of Cases	I	II	III	I	II	III	I	II	III		Without Miotics	With Pilocarpine	Raised	Unaltered	Increased	Decreased	Unaltered	Increased	Decreased
1	..	..	1	..	..	1	..	..	1	6	1	..	..	..	..	1	..	..	1
2	1	..	..	..	1	..	..	1	..	7	2	..	..	..	1	..	2	..	..
	1	..	..	..	..	1	..	1	..										
8	2	..	..	2	..	..	2	..	..	8	2	..	..	2	2	..	2	..	..
	1	..	..	1	..	..	..	1	..		1	..	..	1	..	..	1	..	..
	1	..	..	..	1	..	..	..	1		1	..	..	1	..	..	1	..	..
	1	..	..	..	1	..	..	..	1		1	..	..	1	..	..	..	1	..
	..	1	..	..	..	1	1	..	..		1	..	..	1	..	..	..	1	..
	1	..	..	..	1	..	..	1	..		1	..	..	1	..	..	1	..	..
	..	..	1	..	..	1	..	..	1		1	..	..	1	..	..	..	..	..
5	..	..	1	..	..	1	..	..	1	9	..	1	..	..	1	..	1	..	..
	..	1	..	..	1	..	1	..	..		..	1	..	1	..	..	1	..	..
	..	1	..	..	1	..	..	1	..		1	..	..	1	..	..	..	..	1
	..	1	..	..	1	..	..	1	..		1	..	..	1	..	..	1	..	..
	..	1	..	..	1	..	..	..	1		1	..	..	1	..	..	..	1	..
2	..	1	..	1	..	..	1	..	..	10	1	..	..	1	..	..	1	..	..
	1	..	..	..	1	..	..	..	1		1	..	..	1	..	..	1	..	..
3	..	1	..	..	..	1	..	..	1	11	..	..	1	..	1	..	1	..	..
	..	1	..	..	..	1	..	..	1		1	..	..	1	..	..	1	..	..
	..	1	..	..	1	..	..	1	..		1	..	..	1	..	..	1	..	..
11	1	..	..	..	1	..	1	..	..	12	..	..	1	..	..	1	..	..	1
	1	..	..	1	..	..	1	..	..		1	..	..	1	..	..	1	..	..
	1	..	..	..	1	..	1	..	..		1	..	..	..	..	1	1	..	..
	..	1	..	..	1	..	..	1	..		1	..	..	..	..	1	1	..	..
	1	..	..	..	..	1	..	..	1		1	..	..	..	1	..	1	..	..
	..	1	..	..	1	..	..	1	..		1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..		1	..	..	1	..	..	1	..	..
	..	1	..	..	..	1	..	..	1		1	..	..	..	..	1	..	..	1
	1	..	..	1	..	..	1	..	..		1	..	..	1	..	..	1	..	..
	..	1	..	..	..	1	..	..	1		1	..	..	1	..	..	1	..	..
	..	..	1	..	..	1	..	..	1		..	1	..	1	..	..	1	..	..
4	..	1	..	..	..	1	..	1	..	13	1	..	..	1	..	..	1	..	..
	..	1	..	1	..	..	1	..	..		..	1	..	..	..	1	..	..	1
	..	1	..	..	..	1	..	..	1		1	..	..	..	1	..	..	1	..
	..	1	..	1	..	..	..	..	1		1	..	..	..	..	1	..	..	1
3	1	..	..	1	..	..	..	1	..	14	1	..	..	1	..	..	1	..	..
	..	..	1	..	..	1	..	..	1		1	..	..	..	1	..	1	..	..
	1	..	..	..	1	..	1	..	..		1	..	..	..	1	..	1	..	..
1	..	1	..	1	..	..	1	..	..	15	1	..	..	..	..	1	1	..	..
1	1	..	..	..	1	..	..	1	..	16	1	..	..	1	..	..	1	..	..

3	2	..	..	2	..	..	2	..	..
	..	1	..	..	1	..	1	..	..
1	1	..	..	1	..	..	1	..	..
2	1	..	..	..	1	..	..	1	..
	1	..	..	..	..	1	1	..	..
2	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	..	1	..
2	1	..	..	..	1	..	..	1	..
	1	..	..	1	..	..	1	..	..
1	..	1	..	1	..	..	1	..	..
4	..	1	..	1	..	..	..	1	..
	..	1	..	1	..	..	1	..	..
	2	..	..	2	..	..	2	..	..
4	..	1	..	1	..	..	1	..	..
	1	..	..	..	1	..	..	..	1
	..	1	..	..	1	..	..	1	..
	..	..	1	..	1	..	..	1	..
1	1	..	..	..	1	..	1	..	..
1	..	1	..	..	1	..	..	1	..
2	2	..	..	2	..	..	2	..	..
1	1	..	..	1	..	..	1	..	..
4	..	1	..	..	1	..	..	1	..
	..	1	..	..	1	..	..	..	1
	..	..	1	..	1	..	..	1	..
	..	..	1	..	..	1	..	..	1
2	..	1	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
1	..	..	1	1	..	..	..	..	1
1	..	..	1	1	..	..	1	..	..
3	..	1	..	..	1	..	1	..	..
	1	..	..	1	..	..	1	..	..
	..	..	1	..	1	..	..	1	..
1	..	1	..	1	..	..	..	..	1
1	..	1	..	1	..	..	1	..	..
4	3	..	..	3	..	..	3	..	..
	..	1	..	..	..	1	..	..	1
2	..	1	..	..	1	..	..	..	1
	1	..	..	1	..	..	1	..	..
3	2	..	..	2	..	..	2	..	..
	..	1	..	..	1	..	..	1	..
2	1	..	..	1	..	..	1	..	..
	..	1	..	1	..	..	..	1	..
1	1	..	..	1	..	..	1	..	..
1	1	..	..	..	1	..	..	1	..

17	2	..	..	2	..	..	2	..	..
	1	..	..	1	..	..	1	..	..
18	1	..	..	1	..	..	1	..	..
19	..	1	..	..	..	1	..	..	1
	1	..	..	..	1	..	1	..	..
20	1	..	..	1	..	..	1	..	..
	1	..	..	..	1	..	1	..	..
22	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
23	1	..	..	1	..	..	1	..	..
24	..	..	1	..	..	1	..	..	1
	1	..	..	1	..	..	1	..	..
	2	..	..	2	..	..	2	..	..
25	1	..	..	..	..	1	..	..	1
	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
	..	..	1	..	..	1	..	..	1
26	..	..	1	1	..	..	..	..	1
28	1	..	..	1	..	..	1	..	..
30	2	..	..	..	..	2	..	..	2
31	1	..	..	..	1	..	1	..	..
36	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
	1	..	..	..	1	..	1	..	..
	1	..	..	1	..	..	1	..	..
40	..	..	1	..	..	1	..	..	1
	1	..	..	1	..	..	1	..	..
41	1	..	..	..	1	..	1	..	..
43	..	..	1	..	..	1	..	..	1
44	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
45	..	1	..	1	..	..	1	..	..
46	1	..	..	1	..	..	1	..	..
47	3	..	..	3	..	..	3	..	..
	1	..	..	1	..	..	..	1	..
48	1	..	..	..	1	..	..	1	..
	..	..	1	..	1	..	..	1	..
49	2	..	..	2	..	..	2	..	..
	1	..	..	1	..	..	..	1	..
50	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	1	..	..
51	1	..	..	1	..	..	1	..	..
60	1	..	..	1	..	..	1	..	..

TABLE 3.—*Observations on One Hundred and Twenty-Two Cases Before and After Iridencleisis by Holth's Method—Continued*

	Before Operation									After Operation									
	Tension			Vision			Field of Vision			Reexamination, Number Months After Operation	Tension			Vision			Field of Vision		
											Normal								
No. of Cases	I	II	III	I	II	III	I	II	III		Without Miotics	With Pilocarpine	Raised	Unaltered	Increased	Decreased	Unaltered	Increased	Decreased
1	..	..	1	..	1	..	..	..	1	61	1	..	..	1	..	..	1	..	..
1	..	1	..	1	..	..	1	..	..	62	1	..	..	1	..	..	1	..	..
3	..	..	1	..	..	1	..	..	1	65	..	..	1	1	..	..	1	..	..
	..	1	..	1	..	..	1	..	..		1	..	..	1	..	..	1	..	..
	..	1	..	1	..	..	1	..	..		..	..	1	..	..	1	..	..	1
1	..	..	1	1	..	..	1	..	..	69	1	..	..	1	..	..	1	..	..
1	..	1	..	1	..	..	..	1	..	70	..	..	1	..	..	1	..	..	1
2	..	1	..	..	1	..	..	1	..	72	1	..	..	..	..	1	1	..	..
	1	..	..	1	..	..	1	..	..		1	..	..	1	..	..	1	..	..
1	..	1	..	1	..	..	..	1	..	75	..	..	1	..	.	1	..	..	1
2	2	..	..	2	..	..	2	..	..	76	2	..	..	2	..	..	2	..	..
2	..	1	..	..	1	..	..	1	..	82	1	..	..	1	..	..	1	..	..
	1	..	..	1	..	..	..	..	1		1	..	..	1	..	..	1	..	..
2	1	..	..	1	..	..	..	..	1	84	..	..	1	..	..	1	..	..	1
	1	..	..	1	..	..	1	..	..		..	..	1	..	..	1	..	..	1
1	..	1	..	..	1	..	..	1	..	86	1	..	..	1	..	..	1	..	..
3	1	..	..	..	1	..	1	..	..	89	1	..	..	1	..	..	1	..	..
	..	1	..	1	..	..	..	1	..		1	..	..	1	..	..	1	..	..
	..	..	1	1	..	..	..	1	..		1	..	..	1	..	..	1	..	..
1	..	..	1	..	1	.	..	..	1	92	1	..	..	1	..	..	1	..	..
1	1	..	..	..	1	..	1	..	..	99	..	1	..	1	..	..	1	..	..
1	1	..	..	1	..	..	1	..	..	106	1	..	..	..	..	1	..	..	1
1	1	..	..	1	..	..	1	..	..	107	..	..	1	..	1	..	..	..	1
1	1	..	..	1	..	..	1	..	..	109	..	1	..	..	..	1	1	..	1
2	1	..	..	1	..	..	1	..	..	113	..	1	..	..	..	1	..	..	..
	1	..	..	1	..	..	1	..	..		..	1	..	..	1	..	1	..	..
1	1	..	..	1	..	..	1	..	..	125	1	..	..	1	..	..	1	..	..
1	..	..	1	..	..	1	..	..	1	129	1	..	..	1	..	..	1	..	..
1	1	..	..	1	..	..	1	..	..	132	1	..	..	1	..	..	1	..	..
1	..	..	1	1	..	..	..	..	1	159	1	..	..	1	..	..	1	..	..
122	60	44	18	64	39	19	60	32	30		97	10	15	80	17	25	91	8	23
	122			122			122				122			122			122		

As the numbers are too small, it is aimless to undertake a percentile analysis of the single columns of table 3. This would not yield a true picture. However, it is evident that iridencleisis is suitable not only in those cases of glaucoma in which intra-ocular pressure is slightly increased, vision good and the visual field practically unaltered, but even in cases in which the tension is high and vision and the visual field are both poor.

I shall try to demonstrate my opinion by a few figures: Sixty eyes were operated on that had tension I (from 5.5/3 + to 5.5/1), vision I (from 6/5 to 6/12) and visual field I (either a normal field or one in which the shrinkage had not reached the 20 degree circle away from the periphery in any of the eight chief meridians). In 28 (48.7 per cent) at reexamination the tension was normal without miotics and the vision and visual fields were unaltered. In 4 other cases the raised tension could be normalized with pilocarpine. In 2 of the 4 the fields and vision were unaltered.

On the other hand, of the 18 patients whose tension was so greatly increased that it had to be classified under category III, that is, 10/3 +, it was normalized in less than 13 without miotics. In 4 others it could be kept normal with drops. In only 1 case did the operation have no regulating effect on the pressure.

With respect to the visual fields and vision most of the patients in the last-mentioned group endured the operation very well. The field was lost in only 1 of the 30 cases in which it had shrunk inside the 20 degree circle from the fixation point. In this case the tension was 10/2, or about 60 mm. of mercury, and the vision was very poor. In another case before operation the tension was 7.5/1 and vision 6/18, but the field extended only 3 degrees from the point of fixation on the nasal side. Reexamination forty-five months after iridencleisis showed the same vision and visual field, but the pressure was normalized. My longest observed case showed a tension of 10/3 + (about 55 mm. of mercury) field III and vision I before operation. One hundred and fifty-nine months afterward the pressure without pilocarpine was 5.5/2, or about 30 mm. of mercury, but with miotics it was 5.5/4. Vision and field were unaltered.

It may be objected that it is not fair to include those cases in which one is forced to keep the tension normal with miotics. I emphasize that I do it because there are only 10 of these cases, and they all showed a very good result from operation.

The results for vision show that this, as a rule, is not affected unfavorably when it is fairly good before operation.

There may be a transient small decrease caused by postoperative astigmatism, but this is slight, as a rule, when the operator has used

the short peripheral incision with the keratome. Those who use the narrow Graefe knife have to deal with greater and permanent astigmatism. On the other hand, I have occasionally seen a persistent myopia after successful iridencleisis. I cannot give any positive explanation for this phenomenon. As a rule, the myopia seldom exceeds 1.5 to 2 diopters.

SUMMARY

From August, 1911, to Feb. 25, 1931, I performed iridencleisis antiglaucomatosa according to Holth's technic, as my "method of choice" in 152 cases of chronic glaucoma. One hundred and twenty-two of these patients were reexamined from six to one hundred and fifty-nine months after the operation. Twenty of them underwent either total iridectomy and inclusion subconjunctivally of one or both iridal pillars, or peripheral iridectomy with inclusion of the iris in the wound. Iridencleisis was not followed by reoperation, but was used in 3 cases in which other operations had been tried before without result.

In all cases iridencleisis was performed with the 6 mm. bent stop keratome designed by Holth. Scissors were never employed in forming the subconjunctival tunnel. I think that this technic is of the greatest importance for producing the subconjunctival fistula. It also appears that meridional iridotomy is a superior method for decreasing tension. None of the fistula-forming material is lost, the pupil is but slightly dislocated, and dazzling of the eye is avoided.

In only 3 cases did the operation have a direct detrimental effect on the eyes. In the rest of the not quite successful cases (22), it distinctly delayed the inevitable catastrophe.

Duverger's retrobulbar injection greatly diminishes the chance of a dangerous hemorrhage during the emptying of the anterior chamber. A slight massage through the closed lids from the third to the fifth day after the operation contributes to the building of the filtration scar.

No conclusions can be drawn regarding the filtrating power of the scar from its appearance. Real outward fistulating scars are rare in iridencleisis performed lege artis. Thus the phenomenon of Seidel is usually negative. When the operation is performed properly, no thin bleb is formed just at the limbus over the included iris. The latter is covered with relatively thick and well vascularized conjunctiva as well as by the superior scleral beak. The ciliary body is covered by the inferior scleral beak. Late infection is practically never seen for that reason and also because the ciliary body is never exposed in the wound as it often is in Elliot's trephining.

Iridencleisis offers no more security for a lasting good result than other operations for glaucoma. In 2 cases I found a good result fifty and seventy-five months, respectively, after operation, but at reexami-

nation one hundred and thirty-two and one hundred and two months after iridencleisis the final result was poor. In 2 other cases the result at first was good, and then for no visible reason became poor.

I believe, however, that when a delay in the progress of the glaucoma is obtained for thirty-six months there is a chance of lasting cure. I think that I have secured a favorable result in 83.5 per cent of my reexamined patients, a figure obtained by averaging the results for tension, vision and field of vision. In 87.7 per cent of cases the tension was normalized, in 79.5 per cent vision was unaltered or even increased and in 83.2 per cent the field of vision was the same or increased.

Just as in other operations for glaucoma, the best results are obtained when operation is performed early, I think that Holth's iridencleisis, especially when it is combined with meridional iridotomy, gives a relatively certain and lasting result without exposing the eye to considerable risk later.

THE PRESENCE OF VITAMIN A IN THE RETINA*

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In the course of a series of investigations on the structure and composition of the fluids and tissues of the eye in which I and my co-workers [1] have been engaged for some time, attention was directed to the possible occurrence of vitamin A in the retinal tissue. The technic employed was essentially that which has become familiar in recent years to students of nutrition.

PLAN OF INVESTIGATION

Preparation of Retinal Tissue.—Fresh ocular tissue procured at the abattoir was brought to the laboratory, where all the fat and adherent tissues were cut away, the sclera being left clean and dry. The eyeball was then cut in two with a sharp razor blade. The halves were inverted and the vitreous peeled from the retina. The retinal tissue was then removed from the choroidal layer. A few strands of choroidal pigment were found adherent to the retina; hence the latter was gently moved through distilled water to remove the adherent pigment.

The retinal tissue was placed in petri dishes and frozen solid by means of dry ice (solid carbon dioxide); it was immediately placed in a special Hempel vacuum desiccator. Within twelve hours, the tissue was moisture-free. It was removed, comminuted in a mortar and returned to the vacuum desiccator to complete the drying. No effort was made to remove the last traces of water. The dried retinas were placed in a colored bottle which was then filled with carbon dioxide and stoppered. The tissue was thus preserved during the whole experimental period of almost five months without apparent loss of potency. One hundred hog eyes furnished approximately 1.2 Gm. of dried substance, representing about 4 per cent of the fresh retinal tissue. Chor-

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* The expense of this experimentation was defrayed, in part, by a grant from the Committee on Scientific Research of the American Medical Association.

1. Yudkin, A. M.; Kriss, M., and Smith, A. H.: Am. J. Physiol. **97**:611 (July) 1931.

oidal tissue was likewise removed from the eye after the retina was stripped off. It was treated and preserved in the same manner as the retinal tissue.

Experimental Animals.—Young white rats, free from any visible disease, varying in age from 21 to 25 days and weighing from 40 to 45 Gm. were used. The stock diet received by the mother during pregnancy and by the litter during the period of lactation until the time of weaning consisted of calf meal, as recommended by Maynard.[2] In addition, the animals were fed approximately 0.5 Gm. of liver daily. One group of animals, procured from the Connecticut Agricultural Station, received 3 per cent of cod liver oil with the stock diet.

Diets for the Feeding Experiments.—The basal diet devoid of vitamin A was prepared according to the directions followed in Prof. L. B. Mendel's laboratory:

	Per Cent, Gm.
Extracted casein	18
Starch	63
Dehydrogenated fat (Crisco)	15
Salts (Osborne and Mendel)	4
Dried yeast (irradiated) daily	0.4

On this diet the rats failed to grow well after about twenty-one days, and ocular disturbance usually developed a few days after the decline of weight.

When the untoward symptoms were well marked, supplementary feeding of retinal material was undertaken each day with the purpose of ascertaining whether the material tested contained vitamin A which would manifest itself with a relief of the ocular disturbance and resumption of growth.

EXPERIMENTAL FINDINGS

SERIES 1.—Typical results of the feeding tests with a group of animals are shown in chart 1, in which rat C represents the characteristic gain of body weight on the mixed diet advocated by Smith and Bing.[3] Rat 3 represents the failure on the basal diet, without vitamin A. The ophthalmia appeared on the twenty-first day and grew progressively worse, the animal dying on the fifty-fourth day. Autopsy revealed collections of pus at the base of the tongue and in the nasal sinuses and inner ears. The abdominal wall and viscera were almost devoid of fat. The harderian gland was considerably shrunken and infected. Rat 1 was maintained for twenty-five days on the same basal diet until the ophthalmia was marked. Thereafter it received a daily

2. Miller, R. C., and Maynard, L. A.: Am. J. Physiol. **79**:626, 1927.
3. Smith, A. H., and Bing, F. C.: J. Nutrition **1**:179, 1928.

supplement of 50 mg. of dried retinal tissue; complete recovery and resumption of growth occurred. It is evident, therefore, that this quantity of dried retinal tissue is adequate to supply the vitamin A requirement of the growing animal.

SERIES 2.— Chart 2 shows the outcome of feeding tests intended primarily to ascertain whether smaller quantities of dried retinal tissue would be adequate as sources of the essential vitamin A. The graph of rat 6 illustrates the effect of the basal diet without the added vitamin. The animal died with the characteristic signs of vitamin A deficiency.

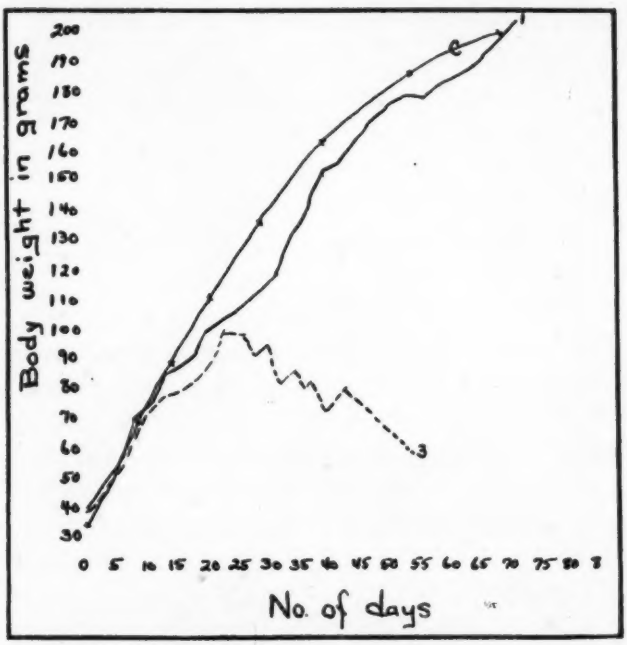

Chart 1 (series 1).—Typical results of feeding tests. *C*, female rat showing gain of weight on mixed diet; *1*, female rat fed 50 mg. of retina; *3*, negative control rat, female.

The record of rat C illustrates the rate of gain on a mixed diet, to serve for comparison as an example of normal growth. Rats 9 and 12 were maintained on basal diets until symptoms of deficiency appeared on the thirty-third day, whereupon they received respectively 20 and 30 mg. of dried retinal tissue, as indicated on chart 2. Recovery followed and growth was resumed, though at not quite the "normal" rate. Rat 8, similarly receiving 100 mg. of butter-fat daily, did not grow better, so that it may be inferred that the retinal tissue studied was approximately four times as potent as butter-fat as a source of vitamin A.

SERIES 3.—The record of rat C on a mixed diet serves for comparison as an index of the normal rate of growth. The other animals were kept on the basal diet until they showed symptoms of a vitamin A deficiency. Thereupon they received dried retinal tissue daily in different amounts as indicated. It is evident that 10 mg. per day was inadequate. With the larger quantities satisfactory growth was secured. From this experiment it can be seen that the recuperative celerity depends on the amount of retinal tissue supplied.

SERIES 4.—These experiments were conducted in a manner comparable to those already described and are self-explanatory. The

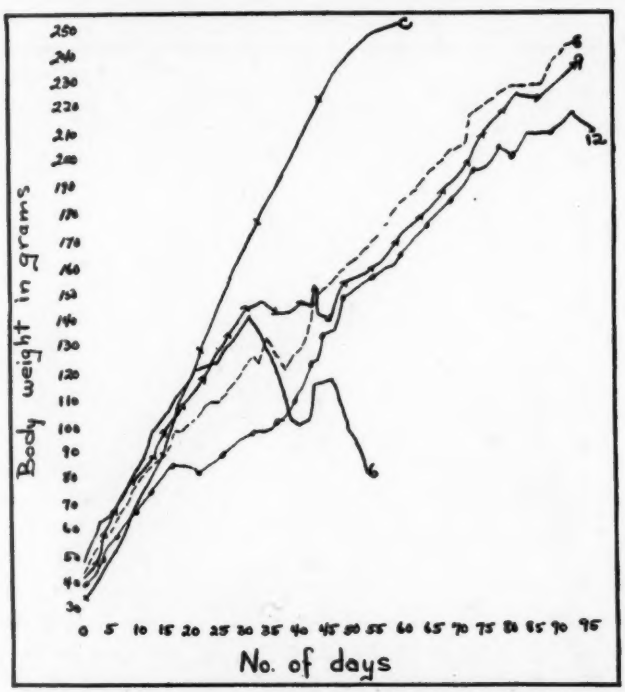

Chart 2 (series 2).—Results of feeding tests on female rats with smaller quantities of retinal tissue. *C*, normal control rat; *6*, negative control rat; *8*, rat given 100 mg. of butter-fat; *9*, rat given 20 mg. of retina; *12*, rat given 30 mg. of retina.

poverty of the choroidal tissue in vitamin A, as contrasted with the retina, is obvious.

SERIES 5.—In this group of experimental animals, rats 26 and 27 were supplied with supplementary sources of vitamin A (in addition to the basal diet) from the beginning of the test. It will be observed that under these conditions, when no preliminary depletion of the animals' store of vitamin A had been attempted, as little as 20 mg. of dried retinal tissue per day sufficed to permit satisfactory growth.

In rats 33 and 34, which were depleted of their vitamin A storage on the basal diet before the supplementary feeding was begun, the poverty of the choroidal tissue compared with retinal tissue as a source of vitamin A is manifest.

Chemical Characteristics of Retinal Tissue.—The retinal tissue, freed from moisture, showed approximately 20.3 per cent of substance extractable with ether. The extract was a fatty, yellowish substance which gave a characteristic violet blue color when the arsenic trichloride test was applied to it. If this test, devised by Rosenheim,[4] is significant of the presence of vitamin A, the extract must contain it. Treatment of the residual tissue with hot alcohol gave approximately 18 per cent

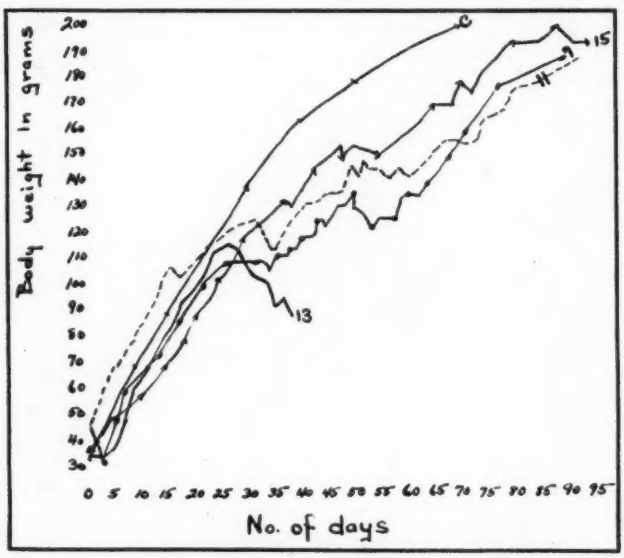

Chart 3 (series 3).—Effect of retinal tissue on growth in female rats with vitamin deficiency as a result of the basal diet. *C*, rat showing normal growth; *7*, rat given 30 mg. of retina; *11*, rat given 20 mg.; *13*, rat given 10 mg., and *15*, rat given 50 mg.

extractive substance. This also was fatty and yellowish. The vitamin A test on this substance did not give a positive reaction. The retinal tissue, free from moisture, contained about 12 per cent of nitrogen.

COMMENT

The ocular changes that accompany the loss of weight in these animals on a faulty diet have been observed by practically all investigators. In the early stages the ocular lesion is known as xerophthalmia,

4. Rosenheim, C., and Drummond, J. C.: Biochem. J. **19**:753, 1925.

and in the more advanced stages as keratomalacia. In addition to the corneal and glandular disturbance Holm [5] and Sugita [6] noted a diminution of visual acuity when the experimental animal, fed on the deficiency diet, was placed in a dark room after being exposed to the sunlight. They called this manifestation hemeralopia.

Parallel ocular disturbances have been found in man, and they seem to be related to a deficiency of vitamin A in the diet. Evidence of this functional hemeralopia has also been observed in various parts of the world. Striking examples were noted by Aykroyd [7] in his study

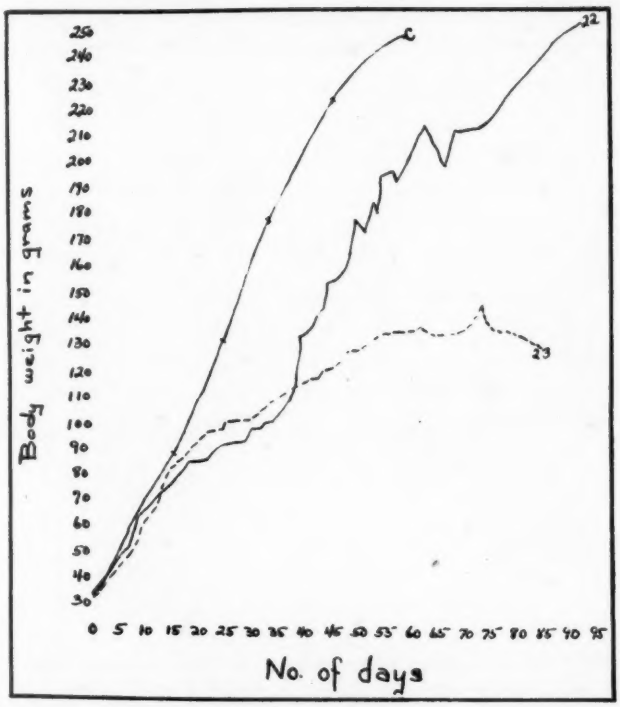

Chart 4 (series 4).—Effects of tests on male rats. *C*, normal control rat; *22*, rat given 50 mg. of retina, and *23*, rat given 50 mg. of choroidal tissue.

of the people of Newfoundland and Labrador. He reported an interesting phase of this disturbance in a person who had learned that dark glasses protected him from the disease; thus covering one eye up by day insured the use of one eye at night. One or two doses of cod liver oil cleared up the condition in from twenty-four to forty-eight hours. Exposure to strong sunlight also played a part; this was true

5. Holm, E.: Acta ophth. **7**:146, 1929.
6. Sugita, Y.: Arch. f. Ophth. **116**:653, 1925.
7. Aykroyd, W. R.: J. Hygiene **30**:357 (Aug.) 1930.

of the Labrador fishermen, who spent a large part of each day in open boats. The disease did not occur in the normal person on a diet of ordinary food, no matter how strongly he might be exposed to sunlight.

In view of the close association of hemeralopia in experimental animals and in man on a diet supposedly deficient in vitamin A, there is reason to believe that vitamin A may play some part in the visual disturbance when the subject is exposed to sunlight.

About fifty years ago Kühne [8] demonstrated a fatty substance in the protoplasm of the retinal epithelial cells which he called lipochrin. In birds, reptiles and fishes he found minute globular bodies in the

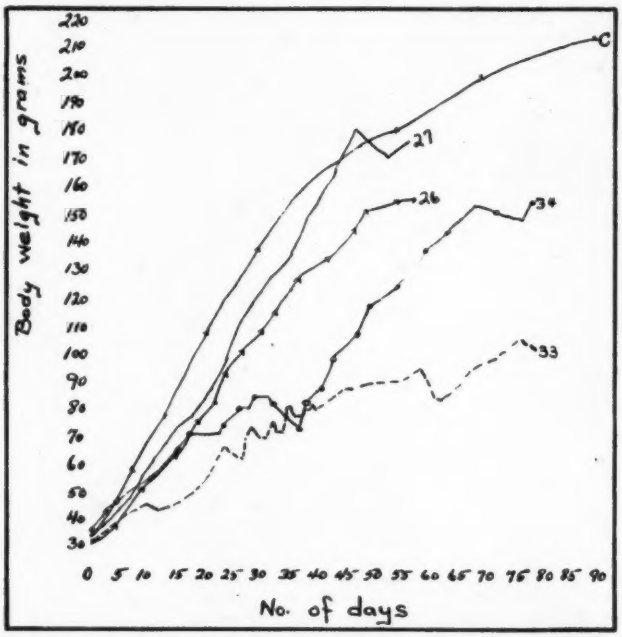

Chart 5 (series 5).—Results of the feeding of retinal and choroidal tissue. *C*, female rate showing normal growth; *26*, female rat, and *27*, male rat given basal diet together with 20 mg. of retina; *33*, female rat given 50 mg. of choroidal tissue, and *34*, female rat given 30 mg. of retina. Rats *33* and *34* had received the basal diet before the supplementary feeding was begun.

inner segment of each cone which, when treated with perosmic acid, turned brown and dissolved readily in fat solvents. These fatlike globules possessed various colors; red and yellow were most frequently met with. Such observations would indicate that the extractive substances that we found are probably located in the external layers of the retinal tissue. The internal layers of the retina are probably rich

8. Kühne, W.: Ztschr. f. Biol. **32**:21, 1895.

in ether and alcohol-extractive substances like the brain tissue, but possess little vitamin A, as has been shown by feeding cerebral tissue to animals on a diet similar to that which I have described in our experiments. If the body is depleted of vitamin A the retina must also lose a good portion of its content of this food factor.

SUMMARY

An examination of the retina of the hog by means of the color reaction of Rosenheim and the feeding of experimental animals depleted of vitamin A has shown the presence of this food factor in the tissue under consideration. Similar findings in the retina of the calf have been reported by Holm.[5] The content of vitamin A in retinal tissue is not inconsiderable. The dried tissue, representing 4 per cent of the retina, is decidedly richer in vitamin A than ordinary butter-fat. Vitamin A is presumably present in the material extractable by ether, which represents at most 20 per cent of the dried retinal tissue. As less than 50 mg. of the latter suffices as a daily source of vitamin A for growing rats on an otherwise adequate diet, it is obvious that the potent principle represents less than 10 mg. of substance, presumably much less than this amount.

On the contrary, the choroidal tissue is either entirely devoid of vitamin A or extremely poor in that factor. Accordingly, it is unlikely that the vitamin A in the retinal tissue is derived from extraneous tissue or contaminating blood.

DIAGNOSTIC SCALES FOR THE 1 DEGREE AND 0.17 DEGREE FORM FIELD STIMULI FOR THE EIGHT PRINCIPAL MERIDIONAL QUADRANTS TAKEN SEPARATELY *

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AND
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In previous papers [1] diagnostic scales were published for the form and color fields based on the area and the average breadth of field in the eight principal meridional quadrants. The average breadth of field was expressed in terms of the distance of the limit in degrees from the center of the field. The area of the field was obtained by plotting the field on a scale of 12 mm. to 10 degrees and measuring the area of the plotted field in square centimeters by means of a planimeter.

Data treated in this way are serviceable primarily in cases in which the contraction produced by the pathologic condition is more or less regular or when an irregular contraction is of sufficient magnitude to affect substantially the area of the field or the average breadth of the field in the eight meridional quadrants.

For the reason that not all irregular contractions are of sufficient magnitude to make a conspicuous change in the area or average breadth of the field, diagnostic comparisons cannot be limited to these features alone. For example, an eye that normally has a large field may, under the influence of a pathologic condition, suffer a considerable contraction over a comparatively small sector without reduction in the area or average breadth of the field below the critical value. In part because of this it has been customary to base the diagnosis on both shape and size of field. However, just as there is a normal range of variation in the size of the field, so there is also a normal range of variation in its

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† From the Research Laboratory of Physiological Optics, Wilmer Ophthalmological Institute, Johns Hopkins Medical School.

‡ From the Graduate School of Medicine, University of Pennsylvania (Dr. Margaret Monroe Smith, Charlotte Eye, Ear and Throat Hospital, Charlotte, N. C.).

1. Ferree, C. E.; Rand, G., and Monroe, M. M.: Studies in Perimetry: 1. Preliminary Work on a Diagnostic Scale for the Form Field, Am. J. Ophth. **9**:95, 1926; 2. Preliminary Work on a Diagnostic Scade for the Color Fields. ibid. **12**: 269, 1929; 4. Preliminary Work on a Diagnostic Scale for the Form Field with a 0.17 Degree Stimulus, ibid. **13**:859, 1930.

shape. For shape, then, as well as for size, a scale or standard of reference is needed for the classification of cases.

While shape cannot conveniently be expressed in numerical terms, scales showing the range and distribution of variation of breadth of field can be formed for as many meridians as are desired. With scales or distribution curves, derived from a large group of normal observers, which show the critical values and the frequency of occurrence of the various areas, the average breadths of field, the breadth in the individual meridians taken separately and perhaps also ratios showing proportionality for different meridians for the same stimulus or for the same meridian for different stimuli, the physician should be aided considerably in deciding whether the various cases that come under his observation should be classed as pathologic, suggestive or nonpathologic. In fact, it is only through the compilation of such data or some equivalent procedure that diagnosis on the basis of test results becomes a reasonable possibility.

It is our purpose in this paper to present diagnostic scales for the limits of the form field in the eight principal meridional quadrants taken separately for stimuli subtending 1 degree and 0.17 degree at the eye. In a later paper similar scales will be given for the limits of the color fields for 1 degree red, green and blue stimuli of the Heidelberg series of pigment papers.

CONDITIONS UNDER WHICH THE WORK WAS DONE

The limits were determined on a Ferree-Rand perimeter with strict observance of all the precautions prescribed for the use of this perimeter. With this instrument all of the external conditions of the test can be held constant and can be reproduced at will. The illumination at every point in the field was kept constant at 7 foot-candles. The stimuli employed were the white papers from the Hering series of standard papers on a background of the black papers of the same series. The coefficients of reflection of stimulus and background were 78 and 4 per cent, respectively. Two sizes of stimulus were used, 5.8 and 1 mm. At the distance of the arc of the perimeter, 33 cm., these stimuli subtended visual angles of 1 and 0.17 degree. The persons examined included private patients, physicians, nurses, assistants on the hospital staff and a large number of clinical patients. In every instance the refractive condition of the eye was carefully determined and an ophthalmoscopic examination made. No cases showing a pathologic condition, however slight, were included in the series. Ample rest periods were allowed between observations, and care was exercised that no field was taken when the observer was suffering from general fatigue or was otherwise unfit for accurate work. All determinations were carefully checked, but the observer was not given extensive preliminary training. It was not our purpose to make

examinations for the determination of a scale of reference under conditions differing in this respect from those which obtain in the ordinary practice of perimetry. The results may therefore be considered a fair sample of what may be expected in an average group of untrained observers who show no pathologic disturbance.

The limits were determined in eight meridional quadrants (0, 45, 90, 135, 180, 225, 270 and 315 degrees). Two hundred eyes were examined with the 1 degree stimulus and 150 with the 0.17 degree stimulus. The group in which the 1 degree stimulus was used included 75 cases of hyperopia and hyperopic astigmatism, 30 cases of myopia and myopic astigmatism, 40 cases of presbyopia, 5 cases of mixed astigmatism and 50 cases showing no error of refraction or an error no greater than 1 diopter of hyperopia or 0.25 diopter of hyperopic astigmatism. The latter group contained no case of myopia or myopic astigmatism of a detectable amount. For convenience it will be referred to as emmetropic. The observers ranged in age from 8 to 56 years. There were 84 males and 116 females.

The group in which the 0.17 degree stimulus was used included 63 cases of hyperopia and hyperopic astigmatism, 19 cases of myopia and myopic astigmatism, 34 cases of presbyopia, 2 cases of mixed astigmatism and 32 cases, called emmetropic, which showed no error of refraction or no error greater than 1 diopter of hyperopia or 0.25 diopter of hyperopic astigmatism. The range in age was from 11 to 56 years. There were 61 males and 89 females.

RESULTS

A representation of the results is given in charts 1 to 4 and in tables 1 to 4. In charts 1 to 4 the cases are rated on the basis of the value of the limit in degrees in the eight meridional quadrants, respectively. For the purpose of grading and representation in the plot, the cases for each meridian are divided into groups, each group covering a range of 2 degrees. The number of cases in each group is shown on the vertical coordinate; the groups themselves are represented on the horizontal coordinate. The cases are designated with regard to the condition of refraction according to a key shown in the charts.

The results represented in charts 1 and 2 may be analyzed as follows:

1. Considered with reference to either the average breadth of the field or the area of the field in square centimeters, as shown in earlier papers, the emmetropes and hyperopes have the larger fields and the myopes and presbyopes the smaller fields. Considering the meridional quadrants separately, however, this ranking does not hold uniformly, the exception being that in some of the quadrants the breadth for the myopes as a group compares favorably with that for the emmetropes and hyperopes. For the purposes of this paper, therefore, age rather than condition of refraction forms a better basis for grouping.

2. The scatter or range of variation is much greater in all meridional quadrants (with the exception of the 45 degree) for the 0.17 degree than for the 1 degree stimulus. The following reasons are suggested for this fact: (*a*) Smaller differences between individuals can be detected

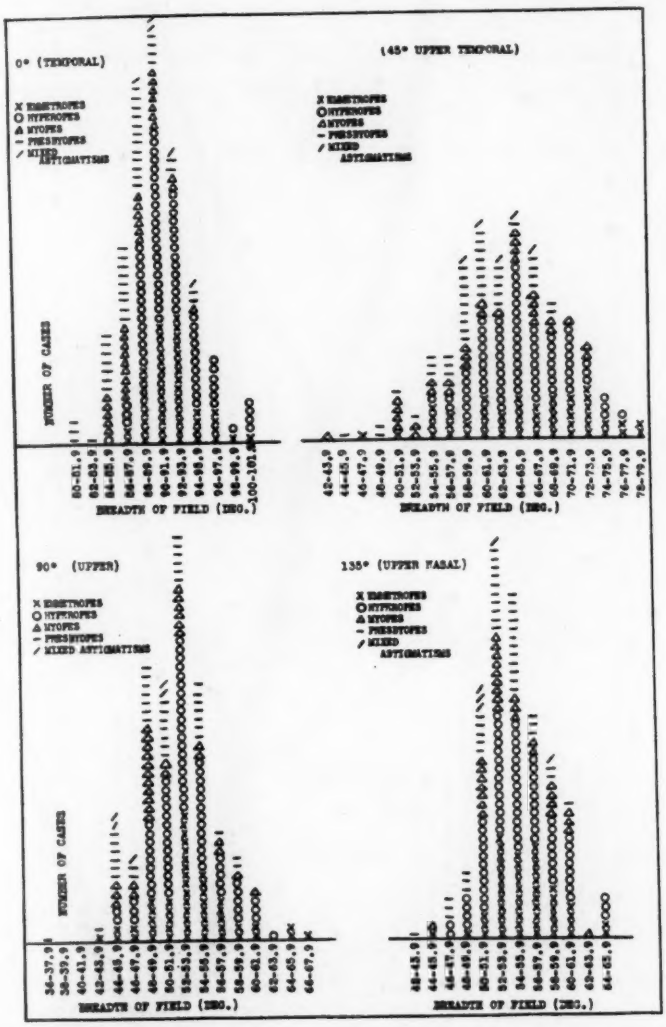

Chart 1.—Limits of the field for the 1 degree form field stimulus in the temporal, upper temporal, upper and upper nasal meridional quadrants: the distribution of 200 nonpathologic cases. The observers were selected to include cases of emmetropia, hyperopia, myopia, presbyopia and mixed astigmatism. The limit of the field in degrees is plotted on the horizontal coordinate and the number of cases on the vertical coordinate.

with the 0.17 degree stimulus; that is, the stimulus of the lower visibility is more sensitive for the detection of this as well as the other

factors and variants which influence the limits of sensitivity. (*b*) The results with the smaller stimulus are more strongly affected by individual differences in refractive condition and in the space sensitivity of the retina. The results with the larger stimulus more nearly give the range

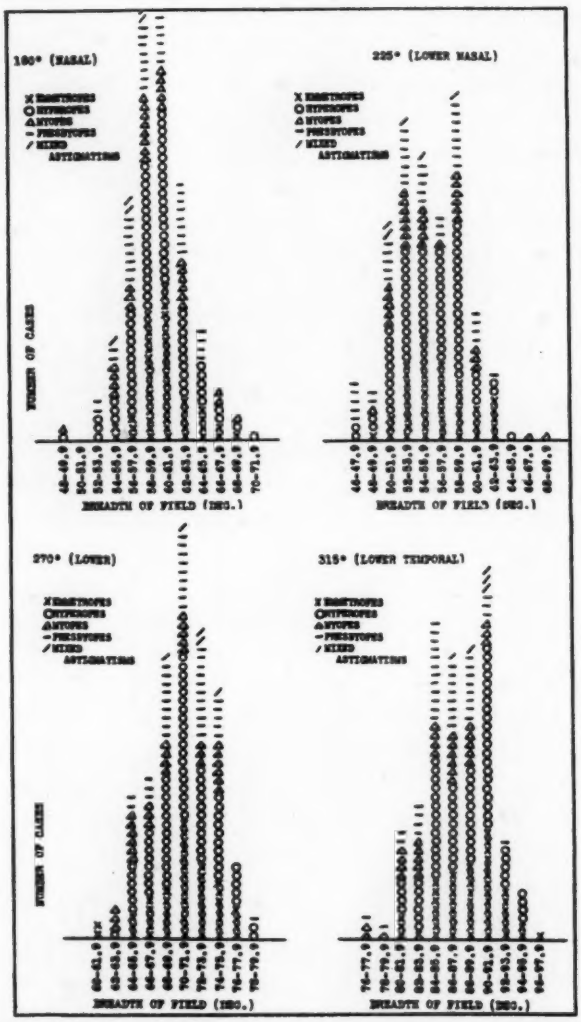

Chart 2.—Limits of the field for the 1 degree form field stimulus in the nasal, lower nasal, lower and lower temporal meridional quadrants: the distribution of 200 nonpathologic cases.

of variation in the sensitivity to light difference. (*c*) The results with the smaller stimulus are more affected by individual differences in size of pupil; that is, size of pupil is an important factor in clearness of imaging, and the small 0.17 degree stimulus requires clearer imaging for

its detection than the larger stimulus. (*d*) The results with the smaller stimulus are also more affected by individual differences in powers of attention and observation than the results with the larger stimulus. Consistency of result with the smaller stimulus, even for the same observer, requires a higher degree of intelligence, training and responsibility than with the larger stimulus.

3. For the 1 degree stimulus the smallest amount of scatter occurs in the 180 degree meridional quadrant and the greatest in the 45 degree

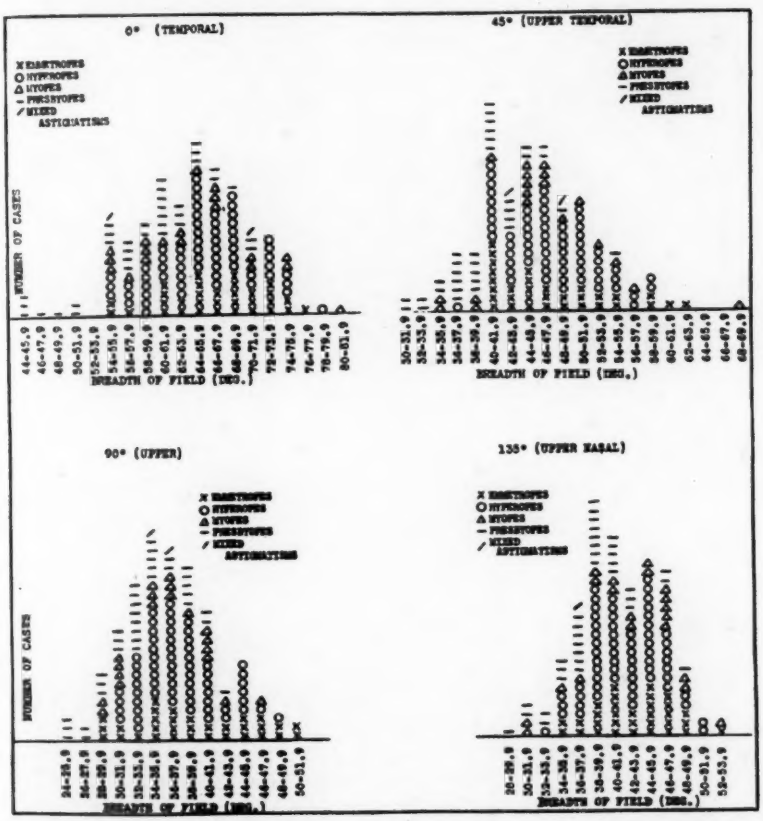

Chart 3.—Limits of the field for the 0.17 degree form field stimulus in the temporal, upper temporal, upper and upper nasal meridional quadrants: the distribution of 150 nonpathologic cases.

meridional quadrant. The remainder, when rated by the median deviation taken as a measure of the scatter, may be divided into two groups. These groups in order from smaller to greater are 0, 135 and 270 degrees, and 90, 225 and 315 degrees. Variability in facial configuration and degree of elevation of the upper lid probably play an important rôle in the unusually large scatter found in the 45 degree meridional quadrant. Stimuli of low visibility, for example, which give limits too

narrow to be affected by these factors do not show an excessive scatter in this quadrant. Because of the excessive amount of scatter, the use of the 1 degree stimulus and other stimuli of high visibility is attended by an unfortunate handicap in this important part of the field in diagnoses based on the limits.[2]

For the 0.17 degree stimulus the ranking from least to greatest scatter is 225 and 135 degrees, 180 degrees, 90 degrees, 270 degrees, 45 degrees, 0, and 315 degrees. For this stimulus the greatest scatter occurs in

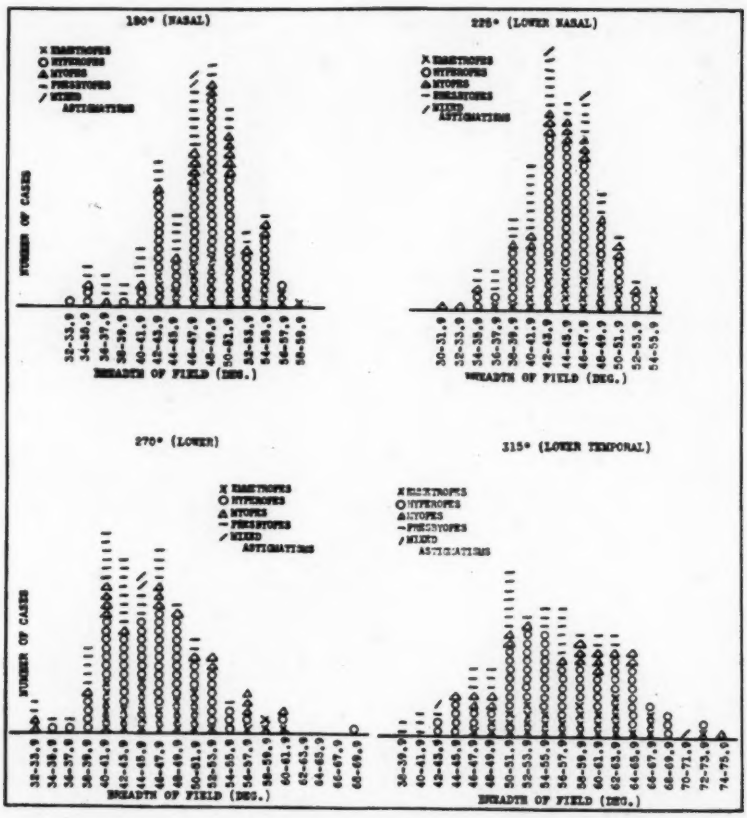

Chart 4.—Limits of the field for the 0.17 degree form field stimulus in the nasal, lower nasal, lower and lower temporal meridional quadrants: the distribution for 150 nonpathologic cases.

general in the regions where the field is widest. Two reasons may be suggested for this: (*a*) Variability in the refractive condition of the eye for the more remote portions of the field. This would especially affect

2. In the collection of these data no extraneous or unusual means were used to secure elevation of the lid. It was deemed unwise to attempt to standardize any interference with the natural position of the lid in the compilation of a scale intended for general use.

small stimuli which require clearer imaging for their discrimination than large stimuli. (*b*) The effect of the sensitivity gradient; that is, in the wider portions of the field sensitivity diminishes more gradually. Individual differences in sensitivity would thus produce greater difference in the location of the limits.

TABLE 1.—*For the 1 Degree Form Stimulus in Each Meridional Quadrant for the Entire Group and for Each Refraction Group: (a) the Limiting Values of the Limit in Degrees; (b) the Average Value; (c) the Median or Middle Value; (d) the Median Deviation expressed in Degrees, and (e) the Median Deviation Expressed as Per Cent of the Median*

Meridional Quadrant, Degrees	Refraction Group	Limiting Values, Degrees	Range, Degrees	Average Limit, Degrees	Median Limit, Degrees	Median Deviation	
						Degrees	Per Cent
0 (Temporal)	Emmetropia....	86.0-100.0	14.0	91.6	91.5	1.5	1.6
	Hyperopia.....	84.0-100.0	16.0	91.7	91.0	2.0	2.2
	Myopia.....	84.0- 95.0	11.0	88.2	88.0	1.5	1.7
	Presbyopia.....	80.0- 95.0	15.0	87.3	87.5	2.5	2.9
	Entire group....	80.0-100.0	20.0	90.3	90.0	2.5	2.8
45 (Upper temporal)	Emmetropia....	47.0- 78.0	31.0	65.4	66.5	5.75	8.6
	Hyperopia.....	55.0- 77.0	22.0	65.0	65.0	3.0	4.6
	Myopia.....	43.0- 72.0	29.0	60.3	60.5	6.5	10.7
	Presbyopia.....	45.0- 68.0	23.0	59.1	59.5	3.0	5.0
	Entire group....	43.0- 78.0	35.0	63.2	63.0	4.5	7.1
90 (Upper)	Emmetropia....	43.0- 67.0	24.0	53.2	52.5	2.75	5.2
	Hyperopia.....	44.0- 62.0	18.0	52.6	53.0	2.5	4.7
	Myopia.....	44.0- 60.0	16.0	50.5	49.0	2.5	5.1
	Presbyopia.....	37.0- 58.0	21.0	50.0	50.0	3.5	7.0
	Entire group....	37.0- 67.0	30.0	51.8	52.0	3.0	5.8
135 (Upper nasal)	Emmetropia....	45.0- 65.0	20.0	55.3	55.0	2.25	4.1
	Hyperopia.....	46.0- 65.0	19.0	54.7	55.0	3.0	5.5
	Myopia.....	45.0- 62.0	17.0	54.1	53.0	3.5	6.6
	Presbyopia.....	42.0- 60.0	18.0	52.5	52.5	2.5	4.8
	Entire group....	42.0- 65.0	23.0	54.3	54.0	2.5	4.6
180 (Nasal)	Emmetropia....	54.0- 68.0	14.0	60.2	60.0	1.75	2.9
	Hyperopia.....	48.0- 70.0	22.0	59.6	60.0	2.5	4.2
	Myopia.....	49.0- 69.0	20.0	59.3	59.0	2.5	4.2
	Presbyopia.....	52.0- 65.0	13.0	59.4	59.5	3.0	5.0
	Entire group....	48.0- 70.0	22.0	59.6	60.0	2.0	3.3
225 (Lower nasal)	Emmetropia....	49.0- 63.0	14.0	56.1	55.5	3.0	5.4
	Hyperopia.....	46.0- 65.0	19.0	55.1	56.0	3.0	5.4
	Myopia.....	48.0- 68.0	20.0	55.4	54.0	3.5	6.5
	Presbyopia.....	46.0- 62.0	16.0	54.0	54.0	4.0	7.4
	Entire group....	46.0- 68.0	22.0	55.4	55.0	3.0	5.5
270 (Lower)	Emmetropia....	60.0- 77.0	17.0	70.4	70.0	2.5	3.6
	Hyperopia.....	62.0- 78.0	16.0	70.0	70.0	2.5	3.6
	Myopia.....	62.0- 75.0	13.0	68.6	68.5	4.0	5.8
	Presbyopia.....	65.0- 74.0	9.0	70.8	71.0	2.0	2.8
	Entire group....	60.0- 78.0	18.0	70.3	71.0	2.5	3.5
315 (Lower temporal)	Emmetropia....	80.0- 97.0	17.0	88.4	88.5	2.5	2.8
	Hyperopia.....	80.0- 95.0	15.0	87.6	88.0	2.5	2.8
	Myopia.....	77.0- 91.0	14.0	84.6	85.0	3.25	3.8
	Presbyopia.....	77.0- 92.0	15.0	85.5	85.5	2.0	2.3
	Entire group....	77.0- 97.0	20.0	87.0	87.0	3.0	3.5

4. A numerical comparison of the relation between meridional quadrants is a valuable feature in the use of scales compiled for the different meridional quadrants. This is particularly true for the 0.17 degree stimulus because of the greater range of variation or scatter of results for this stimulus; that is, fields that were normally wide might suffer

considerable contraction in one or more meridional quadrants and still the limit might remain within the normal range of the group. The abnormality would be revealed, however, by a comparison of the place

TABLE 2.—*For the 0.17 Degree Form Stimulus in Each Meridional Quadrant for the Entire Group and for Each Refraction Group: (a) the Limiting Values of the Limit in Degrees; (b) the Average Value; (c) the Median or Middle Values; (d) the Median Deviation Expressed in Degrees, and (e) the Median Deviation Expressed in Per Cent of the Median*

Meridional Quadrant, Degrees	Refraction Group	Limiting Values, Degrees	Range, Degrees	Average Limit, Degrees	Median Limit, Degrees	Median Deviation	
						Degrees	Per Cent
0 (Temporal)	Emmetropia....	54.0-76.0	22.0	66.3	67.0	4.0	6.0
	Hyperopia.....	55.0-78.0	23.0	65.0	65.0	4.0	6.2
	Myopia.....	55.0-80.0	25.0	64.0	63.0	6.0	9.5
	Presbyopia.....	44.0-70.0	26.0	58.2	60.0	4.75	7.9
	Entire group....	44.0-80.0	36.0	63.6	64.0	4.75	7.4
45 (Upper temporal)	Emmetropia....	40.0-63.0	23.0	47.3	45.5	5.5	12.1
	Hyperopia.....	37.0-58.0	21.0	47.2	47.0	3.5	7.4
	Myopia.....	35.0-68.0	33.0	46.4	45.0	5.0	11.1
	Presbyopia.....	30.0-55.0	25.0	39.5	39.0	3.25	8.3
	Entire group....	30.0-68.0	38.0	45.3	45.0	4.5	10.0
90 (Upper)	Emmetropia....	28.0-51.0	23.0	39.0	38.0	5.5	14.5
	Hyperopia.....	30.0-48.0	18.0	37.3	37.0	3.0	8.1
	Myopia.....	28.0-46.0	18.0	35.7	36.0	5.0	13.9
	Presbyopia.....	25.0-42.0	17.0	33.1	33.0	4.0	12.1
	Entire group....	25.0-51.0	26.0	36.5	36.0	3.5	9.7
135 (Upper nasal)	Emmetropia....	35.0-52.0	17.0	42.8	42.5	4.5	10.6
	Hyperopia.....	32.0-50.0	18.0	41.7	42.0	3.5	8.3
	Myopia.....	30.0-52.0	22.0	41.8	44.0	4.5	10.2
	Presbyopia.....	28.0-48.0	20.0	37.8	38.0	2.5	6.6
	Entire group....	28.0-52.0	24.0	41.0	41.0	3.0	7.3
180 (Nasal)	Emmetropia....	42.0-58.0	16.0	49.3	49.0	3.5	7.1
	Hyperopia.....	33.0-56.0	23.0	47.2	48.0	3.0	6.3
	Myopia.....	35.0-54.0	19.0	46.9	48.0	3.0	6.3
	Presbyopia.....	34.0-54.0	20.0	44.2	44.5	3.5	7.9
	Entire group....	33.0-58.0	25.0	46.9	47.5	3.25	6.8
225 (Lower nasal)	Emmetropia....	37.0-55.0	18.0	45.8	45.5	3.0	6.6
	Hyperopia.....	35.0-53.0	18.0	44.0	44.0	2.0	4.6
	Myopia.....	31.0-53.0	22.0	43.1	44.0	3.5	8.0
	Presbyopia.....	35.0-53.0	18.0	42.1	41.5	3.5	8.4
	Entire group....	31.0-55.0	24.0	43.9	44.0	3.0	6.8
270 (Lower)	Emmetropia....	40.0-60.0	20.0	47.2	46.5	4.0	8.6
	Hyperopia.....	35.0-69.0	34.0	46.5	47.0	3.5	7.4
	Myopia.....	32.0-60.0	28.0	46.2	46.0	6.5	14.1
	Presbyopia.....	32.0-55.0	23.0	42.0	42.0	3.25	7.7
	Entire group....	32.0-69.0	37.0	45.6	45.0	4.0	8.9
315 (Lower temporal)	Emmetropia....	47.0-72.0	25.0	57.6	58.5	5.5	0.4
	Hyperopia.....	42.0-72.0	30.0	56.0	55.0	5.5	10.0
	Myopia.....	45.0-74.0	29.0	56.2	58.0	6.0	10.3
	Presbyopia.....	38.0-62.0	24.0	51.0	50.5	5.0	9.9
	Entire group....	38.0-74.0	36.0	55.2	55.0	5.5	10.0

or ranking of these results in the scale with those obtained in the other meridians. In doubtful cases it is also of great value to compare the results with those of the other eye. This is true also in the use of the scales for the area and average breadth of field.

5. Because of a greater range of scatter of results, the 0.17 degree stimulus is of less value than the 1 degree stimulus for the detection of

abnormalities by an inspection of the results in the various meridional quadrants considered separately. Furthermore it is of no value in cases in which only the nerves that reach the far periphery of the retina are affected. The superiority of it and other stimuli of low visibility, so far as diagnosis by means of the limits is concerned, is in the comparison of the results for the different meridional quadrants of the eye under observation, in a comparison of the results for that eye with those for the other eye, and in cases in which there is a partial loss of sensitivity over a comparatively large area. In the latter cases, stimuli of low visibility show a contraction when stimuli of high visibility show nothing.

6. The frequency distributions more nearly fit the normal or gaussian curve for the large than for the small stimulus. This might be expected from the facts noted in paragraph "2," p. 521. The greatest symmetry of distribution about the median occurs in the horizontal meridian, temporal and nasal. This difference in symmetry in favor of the horizontal meridian is striking for the 1 degree stimulus.

Tables 1 to 4 are intended as a partial analysis of the plots given in charts 1 to 4. In tables 1 and 2 are shown for the total number of cases and for the different refraction groups when each meridional quadrant is taken separately: (*a*) the limiting values of the limit in degrees; (*b*) the average values; (*c*) the median or middle value; (*d*) the median deviation,[3] and (*e*) the per cent the median deviation is of the median. These data are given for the 1 degree stimulus in table 1 and for the 0.17 degree stimulus in table 2. An inspection of the median deviations for the various meridional quadrants offers a numerical comparison of the scatter or dispersion of the individual results for each quadrant.

In tables 3 and 4 are shown for the two sizes of stimulus, respectively, (*a*) the narrowest limit in each of the meridional quadrants; (*b*) the 5 percentile limit (i.e., the value below which lies approximately 5 per cent and above which approximately 95 per cent of the total number of cases), and (*c*) the limit tentatively chosen as suggestive of pathologic disturbance. These are given both for the entire group and for the group excluding presbyopes. They are singled out and featured separately in these tables as an aid to the examiner in selecting a value that he considers differential. While columns 4 and 7 of tables 3 and 4 give our tentative selection of the limits that may be taken to indicate pathologic disturbance, in diagnosis it will be remembered that the relation of the values to each other as well as their absolute magnitude must be taken into account; that is, in a given case in which the greater number of values are large, larger values than we have selected for

3. The median deviation, as a measure of variability or scatter, represents the median or middle of the deviations from the median values in the scale. It specifies a range on either side of the median value within which is included 25 per cent of the total number of the cases.

columns 4 and 7 occurring in one or more of the meridional quadrants may well be considered as warranting suspicion of a period of observation. It may be possible later to formulate a scale showing the normal variation in the relation of the limits to each other for the more significant locations in the field.

At best, even when all the evidence is in, much must be left to the judgment and experience of the examiner. Data collected and presented

TABLE 3.—*For the 1 Degree Form Stimulus in Each Meridional Quadrant for the Entire Group and the Entire Group Excluding Presbyopes: (a) the Narrowest Limit; (b) the 5 Percentile Limit,* and (c) the Limit Tentatively Chosen as Suggestive of Pathologic Disturbance*

	Entire Group			Excluding Presbyopes		
Meridional Quadrant, Degrees	Narrowest Limit, Degrees	5 Percentile Limit, Degress	Limit Suggestive of Pathologic Disturbance, Degrees	Narrowest Limit, Degrees	5 Percentile Limit, Degrees	Limit Suggestive of Pathologic Disturbance, Degrees
0	80	84	80	84	86	84
45	43	51	48	43	53	50
90	37	44	42	43	45	44
135	42	48	42	45	49	44
180	48	54	52	48	54	52
225	46	48	46	46	50	46
270	60	64	60	60	64	60
315	77	80	76	77	80	76

* Largest value in the group which represents the smallest 5 per cent of cases.

TABLE 4.—*For the 0.17 Degree Stimulus in Each Meridional Quadrant for the Entire Group and the Entire Group Excluding Presbyopes: (a) the Narrowest Limit; (b) the 5 Percentile Limit, and (c) the Limit Tentatively Chosen as Suggestive of Pathologic Disturbance*

	Entire Group			Excluding Presbyopes		
Meridional Quadrant, Degrees	Narrowest Limit, Degrees	5 Percentile Limit, Degress	Limit Suggestive of Pathologic Disturbance, Degrees	Narrowest Limit, Degrees	5 Percentile Limit, Degrees	Limit Suggestive of Pathologic Disturbance, Degrees
0	44	52.5	44	54	55	54
45	30	35.0	30	35	40	34
90	25	28.0	24	28	30	28
135	28	33.0	28	30	34	30
180	33	36.0	34	33	39	34
225	31	35.5	34	31	36	34
270	32	36.5	32	32	38	32
315	38	42.5	38	42	45	42

in the form given here are intended only as a guide or aid in making a decision. Any formulation in general terms has to be applied to the individual case with discretion and full consideration of all the attendant factors and circumstances. Moreover, in this connection one fact cannot be emphasized too strongly; namely, such data as we have collected have application only when the controls employed by us in making the examination are used. There is little hope of securing a helpful degree of standardization in diagnosis without the adoption of a standardized procedure of making the examination.

In charts 5 to 8 the analysis of the data is continued graphically. In charts 5 and 6 the points representing the limiting values in each of the meridional quadrants are connected into field maps. It should be remembered, however, that size of field taken as a whole is not the feature stressed in the treatment of the data given in this paper. In charts 7 and 8 the points are not connected to form a field map.

In charts 5 and 6 are represented for the 1 degree and the 0.17 degree stimuli, respectively, the limiting values for the entire group and for the middle 90 per cent of the cases. The 90 per cent group is shown as a shaded zone. Near the middle of this zone a heavy line indicates the position of the median case. Some examiners may attach a great deal of importance to the inner borderline of the upper 95 per

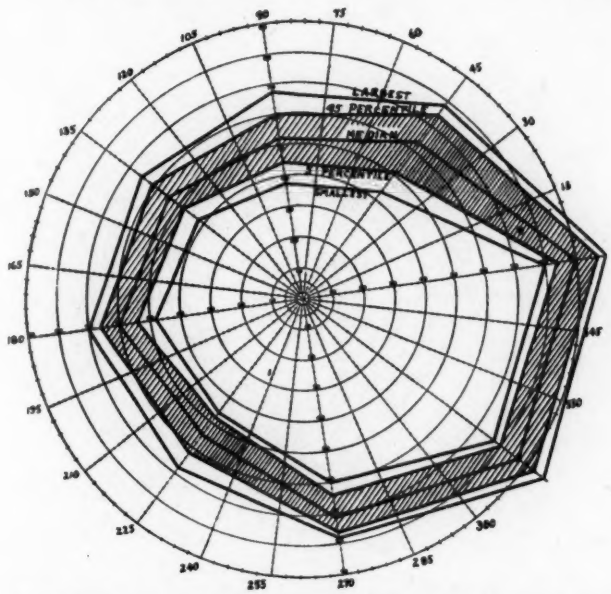

Chart 5.—For the 1 degree form field stimulus the limiting values for the entire group of 200 nonpathologic cases and for the middle 90 per cent of these cases. The 90 per cent group is shown as a shaded zone. Near the middle of this zone a heavy line represents the position of the median case.

cent group. It is helpful to know at least that 95 per cent of a sample nonpathologic group have limits wider than these and only 5 per cent have narrower limits.

In charts 7 and 8 are shown for the 1 degree and 0.17 degree stimuli, respectively, the points that have been selected tentatively as representing critical values for the limits. So far as a wider comparative examination of pathologic and nonpathologic cases under the controls used in our study justifies the selection, cases showing narrower limits than these should be regarded with suspicion. We would prefer, however, that each examiner study the distribution of cases presented and

select for himself the values that he considers critical or differential. In our routine work on clinical cases we have found the scales that we have compiled to be of great value. Without them a decision on the basis of test results would have been impossible in a great number of cases. In early glaucoma, incipient optic atrophy in neurosyphilis, etc., they have been indispensable.

SUMMARY

Scales based on area of field or average breadth of field in the eight principal meridional quadrants are serviceable in diagnosis primarily in cases in which the contraction produced by pathologic conditions is more or less regular or in which an irregular contraction is of sufficient magnitude to affect the area of the field or its average breadth. In all other cases scales for the meridians taken separately are needed for a diagnosis based on the limits of the field. In the foregoing paper such scales have been compiled for the eight principal meridional quadrants (0, 45, 90, 135, 180, 225, 270 and 315 degrees) for the 1 degree and 0.17 degree form stimuli. With reference to these scales, the following points may be noted briefly in summary.

1. Considered with reference to either area of field or average breadth of field, emmetropes and hyperopes have the larger, and myopes and presbyopes the smaller fields. When the meridional quadrants are

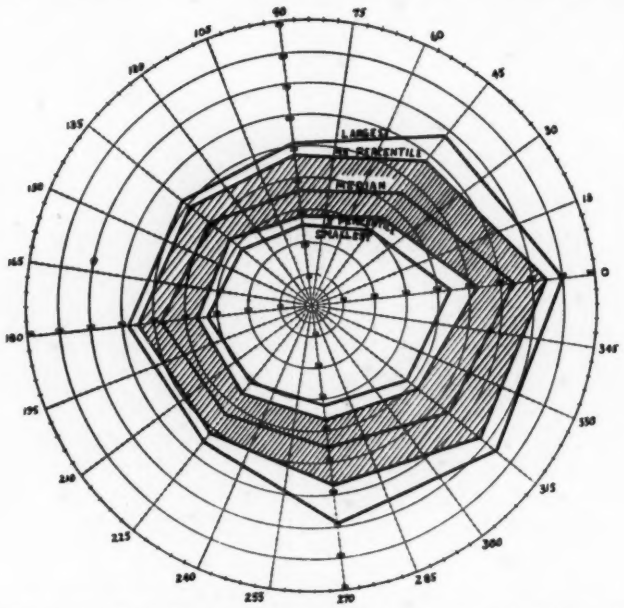

Chart 6.—For the 0.17 degree form field stimulus the limiting values for the entire group of 150 nonpathologic cases and for the middle 90 per cent of these cases. The 90 per cent group is shown as a shaded zone. Near the middle of this zone a heavy line represents the position of the median case.

considered separately, however, this ranking does not hold throughout, i. e., in some of the quadrants the breadth for the myopes taken as a group compared favorably with that for the emmetropes and hyperopes. Age rather than condition of refraction forms a better basis for grouping in scales based on the limits in the individual meridional quadrants.

2. The scatter or range of variation of the results is much greater in all meridional quadrants (with the exception of the 45 degree) for the 0.17 degree stimulus than for the 1 degree stimulus. The following reasons may be suggested for this: (*a*) Smaller differences between individuals can be detected with the 0.17 degree stimulus; that is, a stimulus of low visibility is more sensitive for the detection of these differences as well as the effects of the other factors and variants which influence the

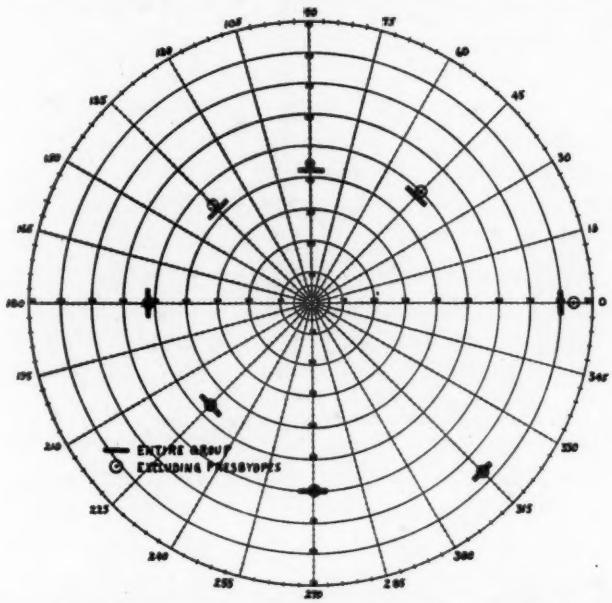

Chart 7.—For each of the eight principal meridional quadrants the point beyond which a contraction of the limit suggests pathologic disturbance. These points are shown for the 1 degree form field stimulus for the entire group and for the group excluding the presbyopes.

limits of sensitivity than stimuli of high visibility. (*b*) The results with the smaller stimulus are more strongly affected by individual differences in the refractive condition of the eye and in the space sensitivity of the retina. The results with the larger stimulus are more nearly exclusively dependent on the range of variation of the function that it is our purpose to test, namely, the sensitivity to light difference. (*c*) The results with the smaller stimulus are more strongly affected by individual differences in size of pupil; that is, size of pupil is an important factor in clearness of imaging, and the small 0.17 degree stimulus requires clearer imaging for its detection than the larger

stimulus. (*d*) The results with the smaller stimulus are also more strongly affected by individual differences in power of attention and observation than results with the larger stimulus. Consistency of result with small stimuli, even for the same observer, requires a higher degree of intelligence, training and responsibility than with large stimuli.

3. For the 1 degree stimulus the smallest amount of scatter occurs in the 180 degree, and the greatest in the 45 degree, meridional quadrant. The remainder, as rated by the median deviation taken as a measure of the scatter, may be divided into two groups. In order from smaller to greater, these groups are: 0, 135 and 270 degrees, and 90, 225 and 315 degrees. Variability in facial configuration and degree of elevation of

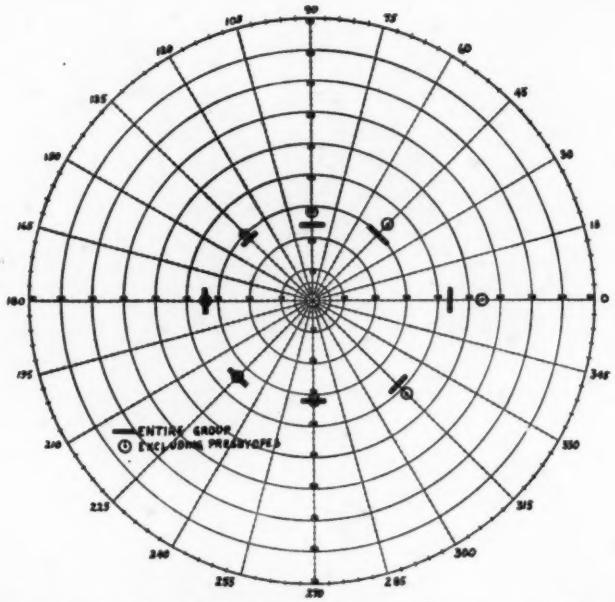

Chart 8.—For each of the eight principal meridional quadrants the point beyond which a contraction of the limit suggests pathologic disturbance. These points are shown for the 0.17 degree form field stimulus for the entire group and for the group excluding the presbyopes.

the upper lid are probably important factors in the excessive amount of scatter found in the 45 degree meridional quadrant. For the 0.17 degree stimulus the ranking as to scatter, in order from least to greatest, is: 225 and 135 degrees, 180 degrees, 90 degrees, 270 degrees, 45 degrees, 0, and 315 degrees. The greatest scatter here occurs in the widest part of the field. This might be expected (*a*) from the effect of variability of the refractive condition in the more remote portions of the field on the discrimination of a small test object, and (*b*) from the greater effect that variations of sensitivity have on the location of the limits in parts of the field where the sensitivity diminishes gradually.

4. A numerical comparison of the relation between meridional quadrants is a valuable feature in the use of scales compiled for the different meridional quadrants. This is particularly true for the 0.17 degree stimulus because of the greater range of variation or scatter of results for this stimulus; that is, fields that were normally wide might suffer considerable contraction in one or more meridional quadrants and still the limit might remain within the normal range of the group. The abnormality would be revealed, however, by a comparison of the place or ranking of these results in the scale with those obtained in the other meridians. In doubtful cases it is also of great value to compare the results with those for the other eye. This is true also in the use of the scales for the area or average breadth of field.

5. Because of a greater range of scatter of results, the 0.17 degree stimulus is of less value than the 1 degree stimulus for the detection of abnormalities on inspection of the results in the various meridional quadrants considered separately. Furthermore, it is of no value in cases in which only the nerve fibers that reach the far periphery of the retina are affected. The superiority of it and other stimuli of low visibility, so far as diagnosis by means of the limits is concerned, is in the comparison of results for the different meridional quadrants of the eye under observation, in a comparison of the results for that eye with those for the other eye, and in cases in which there is a partial loss of sensitivity over a comparatively large area. In the latter cases, stimuli of low visibility show a contraction when stimuli of high visibility show nothing.

6. In tables 3 and 4 are given the values obtained for the narrowest limit of the group, the 5 percentile limit, and the limit that has been tentatively selected by the authors as designating cases that should be regarded with suspicion and kept under observation. For the convenience of the examiner these values have been singled out as aids to diagnosis. The values selected by the authors as designating cases that should be regarded with suspicion, excluding presbyopes, are in the 0, 45, 90, 135, 180, 225, 270 and 315 degree meridional quadrants respectively for the 1 degree stimulus: 84, 50, 44, 44, 52, 46, 60 and 76 degrees; for the 0.17 degree stimulus they are: 54, 34, 28, 30, 34, 34, 32 and 42 degrees.[4]

4. In this connection it may be added that we have not as yet found a nonpathologic case in which the limit for the 1 degree stimulus in the 0 degree (temporal) meridional quadrant was less than 80 degrees if presbyopia was present or 84 degrees if there was no presbyopia. It is interesting to note that in many cases of early glaucoma, the limits in this meridional quadrant range from 70 to 78 degrees. This meridional quadrant is selected for special mention because we have found it to be so frequently affected in very early stages of glaucoma.

6. The frequency of distributions more nearly fits the normal or gaussian curve for the 1 degree than for the 0.17 degree stimuli. This might be expected from the greater tendency toward scatter in the results obtained with the latter stimuli. The greatest symmetry of distribution about the median occurs in the horizontal meridian, temporal and nasal. This difference in symmetry in favor of the horizontal meridian is pronounced and striking for the 1 degree stimulus.

THE RETINAL CHANGES OF ARTERIOSCLEROTIC HEART DISEASE AND ESSENTIAL HYPERTENSION *

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Many papers dealing with the retina in its relation to systemic conditions have appeared in recent years. Likewise numerous wholly descriptive essays have been published concerning retinal changes. However, there have been very few attempts to correlate the modern concepts of heart disease with retinal changes. Even in the latter papers there has been much confusion in the use of such terms as cardiorenal disease, essential hypertension, benign hypertension, arteriosclerosis and arteriosclerotic heart disease. While the descriptions given of the retinal changes are satisfactory, the exact etiology of the underlying conditions is obscurely stated. The newer knowledge of cardiology has apparently escaped the notice of even the most recent authors of essays as well as the authors of the ophthalmologic texts.[1]

The greatest amount of confusion seems to arise as a result of failing to realize that arteriosclerosis and essential hypertension are distinctly different entities. The mere fact that they may occur together has led many observers to refrain from attempting to separate them. From the earliest days of the ophthalmoscope the fundus changes that we now know are hypertensive have been ascribed to arteriosclerosis. Clinical ophthalmoscopy is so much older than sphygomanometry that time has necessarily been required before proper correlation of the observations was obtained.

Let us first consider what is meant by the term arteriosclerotic heart disease. Ordinarily the picture is that of an elderly person, usually over

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* Read by invitation before the Louisville Eye and Ear Society, Dec. 11, 1930.

1. Following the completion of this article there appeared a paper by Fishberg and Oppenheimer on "The Differentiation and Significance of Certain Ophthalmoscopic Pictures in Hypertensive Diseases" (Arch. Int. Med. **46**:901 [Dec.] 1930). They gave a clear description of the hypertensive fundus under the heading "retinal arteriosclerosis and arteriosclerotic retinopathy." The basis for their report was a group of 274 cases.

60 years of age, who presents evidence of either the anginal or the congestive type of heart failure. The palpable arteries are frequently but not always abnormally infiltrated. The blood pressure, which has never been elevated, is normal or lower than normal, regardless of the degree of sclerosis found in the radial vessels. The heart itself is not enlarged and may even be smaller than normal. Electrocardiographic examination will show fairly approximately the amount of myocardial change present. Pathologically the lesions of arteriosclerosis are confined to the arteries and there is no striking involvement of the arterioles.

In hypertensive cardiovascular disease, elevation of the blood pressure is a striking clinical feature. There are many conditions in which the systolic and diastolic blood pressure may be pathologically increased, such as acute and chronic glomerulonephritis, congenital stenosis of the aorta, toxemia of pregnancy, congenital polycystic kidney, arteriovenous aneurysm, lead poisoning, tumors of the suprarenal gland, brain tumors that produce increased intracranial pressure and, finally, essential hypertension. However, in this paper we are limiting our discussion to the hypertensive state that, except for hereditary predisposition, is of unknown etiology. It has been variously termed essential hypertension, hyperpiesia or primary hypertensive cardiovascular disease. Apparently this condition has its beginning in many instances in the early thirties and makes itself manifest in the fifth decade. Often the early cases are found during an examination for life insurance. The blood pressure in the developed case will show a constant systolic level of 150 mm. of mercury or above, with a diastolic level of 100 mm. or above. The heart is definitely enlarged to the left. The electrocardiogram will show alteration first by a shift of the electrical axis to the left (so-called left ventricular predominance), and later by T wave changes. Still later, records showing disturbances of conduction are frequently encountered.

Essential hypertension is a disease of the small vessels. Characteristic lesions have been constantly noted in the arterioles, vessels the size of the afferent and interlobular vessels of the kidney or the branches of the central artery in the retina. The changes apparently consist of hypertrophy of the intima with gradual obliteration of the lumen and hypertrophy of the muscle layer of the media with replacement fibrosis. The arteriolar changes are found throughout all the organs, including the retina. Even in ambulatory patients with essential hypertension, biopsy of the voluntary muscles discloses the same lesions of the arterioles as are found at autopsy.[2] So widespread and pathognomonic is the involvement of the arterioles that some clinicians are inclined to replace the term essential hypertension by that of diffuse vascular disease or

2. Kernohan, J. W.; Anderson, E. W., and Keith, N. M.: Arterioles in Cases of Hypertension, Arch. Int. Med. **44**:395 (Sept.) 1929.

arteriolar sclerotic disease. It is of interest to note that aside from capillary microscopy the retinal arterioles are the only vessels of their size which can be clinically examined.

Sclerotic changes in the larger arteries, such as the radial and brachial vessels, may likewise occur in hypertension. Such lesions are inconstant, bear no relation to the duration of the high blood pressure and are not pathognomonic of the disease in that the sclerosis does not differ from that found in persons without hypertension.

The older concept of arteriosclerosis was that elevation of the blood pressure was always present. There are so many factors involved in the maintenance of normal pressure, some of far more importance than the degree of elasticity of the larger vessels, that change in this one factor does not materially alter the pressure level. On the other hand, in essential hypertension, the extensive sclerosis of the arterioles throughout the body maintains the elevated state of the blood pressure. This arteriolar sclerosis bears no relationship to the degree of involvement of the larger vessels.

Changes of the fundus oculi associated with advanced essential hypertension are readily recognized by the average clinician. The ophthalmologist is needed to detect early and atypical lesions. Since the lesions are frequently progressive, only a composite picture can be portrayed. The arteriolar sclerosis of the retinal vessels is very characteristic. The arterioles are markedly narrowed, having a copper wire appearance; the arterial light reflex is accentuated, giving to the vessels a silver sheath, while the tortuosity and irregularity of lumen are striking. Compression of the veins by the crossed arterioles is considered pathognomonic. Varied stages of papillary and retinal changes are encountered. The disk is red and swollen. Its margins are indistinct, merging with the retina. There may be marked elevation of the disk. Generalized edema of the retina is often found. Scattered hemorrhages and "cotton wool" areas are present. The latter are degenerative spots, fatty and hyaline in nature. Very often these white spots assume a stellate arrangement around the macula.

The retinal picture of the malignant phase of hypertension has been emphasized by Keith, Wagener and Kernohan.[3] There is usually distinct and generalized spastic constriction of the retinal arterioles, in association with generalized edema of the retina, hemorrhages, cotton wool patches and particularly hyperemia and measurable edema of the disk. This papilledema is of special prognostic and diagnostic import.

Should diabetes ensue in a patient with essential hypertension, the ophthalmoscopic observations in the retina are unaltered. The retinitis,

3. Keith, N. M.; Wagener, H. P., and Kernohan, J. W.: The Syndrome of Malignant Hypertension, Arch. Int. Med. **41**:141 (Feb.) 1928.

when present, depends on the severity and stage of the hypertension and not on the coincident diabetes.

The onset of cardiac failure does not alter the retinal changes, except for increased venous distention.

The fundus of simple arteriosclerotic heart disease is in marked contrast. Here the choroidal vessels are chiefly involved, presenting the usual picture of sclerosis. The optic disk is pale. Hemorrhages, transudates and white spots are absent. Only the larger branches of the retinal vessels may show slight narrowing without actual constriction. There is no arteriovenous compression, irregularity of lumen or increase in the light reflex. However, should essential hypertension develop in a person with generalized arteriosclerosis, characteristic retinal vascular sclerosis will develop.[4]

It is important to recognize that the sclerosis of the retinal arterioles and the retinitis that we have described as characteristic of hypertensive heart disease are the result of a present or preexisting elevated state of the blood pressure. Such changes are always indicative of hypertension. They do not reflect sclerosis of the larger arteries which might be simultaneously present. Likewise these lesions bear no relation to varying stages of renal insufficiency which may or may not be present in the same patient. For these reasons the classification of retinitis as given by the ophthalmologic texts is etiologically inaccurate and should be discarded. Such terms as albuminuric retinitis or nephritic retinitis are too comprehensive. There is no such distinct entity as diabetic retinitis since the usual fundus described under this heading cannot be distinguished from similar fundi seen in hypertensive persons with hypertension. The term "arteriosclerotic fundus" is especially confusing. The retinal vascular changes and retinitis occurring in essential hypertension should be referred to as an *arteriolar sclerotic retinitis* and not as arteriosclerotic retinitis. The retinal vessels are arterioles and not arteries.

The incidence of abnormal ophthalmoscopic observations in hypertension varies with the persistence and acuity of the observer. A majority of patients with hypertension of the essential type have some sclerosis of the retinal arterioles, although this sclerosis may be minimal.[5] Further sclerotic changes in the retinal arterioles may be found post mortem although the ophthalmoscope does not disclose any apparent abnormality.[6] In the early stages of hypertensive heart disease, only

4. Pines, N.: Sclerosis of the Retinal Vessels, Brit. J. Ophth. **13**:225, 1929. 1929.

5. Wagener, Henry P.: Retinal Vascular Changes in Hypertension, Ann. Int. Med. **4**:222, 1930.

6. Cohen, Martin: Significance of Pathologic Changes in Fundus in Generalized Arterial and Kidney Diseases, J. A. M. A. **78**:1694 (June 3) 1922.

spasm of the retinal arterioles may be present. We have been fortunate to observe this in a few patients. The usual hypertensive fundus was not present even though the blood pressure was markedly elevated. Spasm was recognized by an irregular constriction along the course of the vessel and was intermittent.

The retinal vascular sclerosis is usually progressive and, once developed, is permanent. The retinal lesions, however, are more evanescent. The areas of degeneration may tend to heal and the hemorrhages disappear. The edema of the disk may diminish. But it is rare for the retina to become entirely normal. Careful examination will always reveal evidence of an old hemorrhage or degenerative spot.

This persistence of the retinal changes resulting from hypertension is of inestimable value in the diagnosis of a type of condition that we designate as "hypertensive heart disease without hypertension."[7] Not infrequently persons are encountered who have cardiac enlargement with normal or low blood pressure but no organic valvular lesions, adhesive pericarditis, severe anemia or other recognized cause for the enlargement. It is our contention that these patients have had a previous hypertension, which explains the cardiac enlargement. Even though the former level of the blood pressure is unknown, convincing clinical or pathologic evidence of a preexisting hypertension can always be found. Aside from the otherwise unexplained cardiac hypertrophy, the patients may show impairment of renal function or uremia, a suggestive roentgenologic silhouette and coronary types of electrocardiograms, such as are so often found in persons known to have hypertension. Autopsy may disclose generalized arteriolar sclerosis, which always evidences a persistent hypertension during the life of the patient. In addition, ophthalmoscopic examination of the fundus may reveal the arteriolar sclerosis and retinitis of known hypertensive heart disease. Since the retinal vascular sclerosis of hypertension will persist even though the blood pressure is temporarily or permanently lowered to normal or subnormal levels,[8] and since these retinal changes are the result only of hypertension, the finding of a hypertensive type of fundus always indicates a present or previously elevated state of the blood pressure. O'Hare and Walker[9] studied the retinas of patients with normal blood pressure who previously had hypertension. They stated that the finding of retinal sclerosis in patients with normal or low blood pressure probably indicates the existence of a previous hyper-

7. Horine, Emmet F., and Weiss, Morris M.: Hypertensive Heart Disease Without Hypertension, Kentucky M. J. **28**:464, 1930.

8. O'Hare, J. P., and Walker, W. G.: Arteriosclerosis and Hypertension, Arch. Int. Med. **33**:343 (March) 1924. Pines, N.: Arterial Hypertension and Retinal Changes, Brit. J. Ophth. **11**:489, 1927.

9. O'Hare and Walker (footnote 8, first reference).

tension with subsequent myocardial weakness or loss of vascular tonus. Altnow [10] confirmed their observations and quoted Fahr who held a similar view. Pines [4] claimed that if one sees marked retinal sclerosis with a normal blood pressure at any age, there may have been essential hypertension in the past and the blood pressure has now become normal, leaving behind permanent changes in the retinal vessels. Wagener [5] said that in his opinion visible sclerosis of the retinal arterioles is practically always evidence of hypertensive disease, whether or not the readings of blood pressure are high at the time of examination. Yater and Wagener [11] stated: "When retinal arteriosclerosis of the hypertension type is found, it always signifies that hypertension is present or has been present. . . . If present in a patient with heart disease, less than sixty years of age, who does not have other evident cause for such disease, hypertension may be assumed to be the cause." Levine and Brown [12] in discussing the etiologic importance of hypertension in coronary thrombosis stated: "Although at the time of the attack the blood pressure readings were normal or low, as a result of finding well marked evidence of retinal sclerosis, it was quite evident that a previous hypertension had existed." In contrast, Friedenwald and Friedenwald [13] described a group of patients with retinal arteriosclerosis without hypertension whose ophthalmoscopic picture did not differ essentially from that in cases of retinal arteriosclerosis with hypertension. Elderly persons presenting this retinal picture have, in general, shown no hypertension, cardiac hypertrophy or other evidence of previous hypertension and no symptoms that would suggest that the blood pressure had fallen below the normal compensated level. Despite their careful clinical studies, we are inclined to believe that this group of patients did have a previous hypertension that caused the retinal changes. Prolonged observation of the patient or examination of the arterioles either by biopsy or at autopsy is necessary before essential hypertension can be excluded.

There are multiple and varied factors that may either temporarily or permanently lower the blood pressure. Any acute infection will result in some fall in arterial pressure, the degree of the reduction in pressure being dependent on the duration and severity of the infection. Chronic infections and wasting diseases likewise cause a lowering of the

10. Altnow, H. O.: Changes in the Eyeground in Vascular Diseases and in Related Conditions, Arch. Int. Med. **40**:757 (Dec.) 1927.

11. Yater, W. M., and Wagener, H. P.: Ophthalmoscopic Signs in Disease of the Heart, Am. J. M. Sc. **178**:105, 1929.

12. Levine, S. A., and Brown, C. L.: Coronary Thrombosis: Its Various Clinical Features, Medicine **8**:254, 1929.

13. Friedenwald, H., and Friedenwald, J. S.: Retinal Blood Vessels in Hypertension and Arteriosclerosis, Bull. Johns Hopkins Hosp. **45**:232, 1929.

pressure. The anemias, exhaustion, starvation, shock and collapse are all factors that lead to hypotension. Hemorrhage, such as nasal or intestinal, will cause a marked fall in pressure. Recently Ayman [14] has called attention to the frequency with which normal blood pressure readings may be found in the early stages of essential hypertension. In hypertensive persons the onset of myocardial failure, uremia or an acute coronary occlusion is a complication that most often produces a marked and often a permanent lowering of the blood pressure. Were such persons examined at the time when the blood pressure was low, the true cause for the cardiac hypertrophy might be considered obscure. However, either the previous or subsequent history or a clinical or pathologic study reveals the true nature of the cardiac enlargement. As we have mentioned, ophthalmoscopic examination of the fundus is an extremely valuable factor in the clinical evidence.

Such alterations in the blood pressure of patients with hypertensive heart disease may explain the confusion that exists in the literature on the correlation of retinal vascular sclerosis with associated clinical states. If the blood pressure is low, the vascular changes should not be considered part of a generalized arteriosclerosis but of a generalized arteriolar sclerosis, and clinical and pathologic evidence of a preexisting hypertension should be sought for in the patient.

There have been demonstrated the same characteristic changes in the arterioles of the voluntary muscles of ambulatory patients with essential hypertension as have been found at autopsy.[2] We are making a similar investigation of the arterioles of the deltoid muscle, obtained by biopsy, in those ambulatory patients whom we designated as having "hypertensive heart disease without hypertension." [15] The preliminary studies reveal the same arteriolar sclerosis as is pathognomonic of known hypertension. We shall thus have additional evidence to support our contention that a previous elevated blood pressure has caused the otherwise unexplained cardiac hypertrophy in this type of case.

The prognostic significance of retinal vascular sclerosis and retinitis has been noted since the early days of ophthalmoscopy. So-called albuminuric retinitis has always had an ominous significance. In general, the prognosis in essential hypertension depends on many varied factors.[16] The persistence of the height of the diastolic level of the blood pressure is of great importance. Varying degrees of myocardial fibrosis, shown

14. Ayman, David: Normal Blood Pressure in Essential Hypertension, J. A. M. A. **94**:1214 (April 19) 1930.

15. Horine, Emmet F.; Weiss, Morris M., and Beard, M. F.: Arteriolar Studies in Patients with Hypertensive Heart Disease Without Hypertension, to be published.

16. Horine, Emmet F.: Prognosis in Rheumatic, Hypertensive and Syphilitic Heart Disease, Am. Heart J. **1**:617, 1926.

electrocardiographically, such as low voltage of the QRS complex, coronary types of T waves, bundle branch block and varying degrees of heart block, forecast progressive heart failure. Renal function should be carefully investigated. The age of the patient and the duration of the hypertension are important. Examination of the fundus is of definite aid in the evaluation of the prognosis. Gradual narrowing of the retinal vessels, indicating progressive arteriolar sclerosis throughout the body, evidences an advancing hypertension. The superposition on simple arteriolar sclerosis of hemorrhages, transudates and other evidences of retinitis is an unfavorable sign. Papilledema is especially significant. Keith, Wagener and Kernohan [3] stressed the prognostic importance of the retinal picture of malignant hypertension. Few of their patients lived more than two years. A cerebral hemorrhage, cardiac failure or uremia caused death in this interval. It is impossible to foretell in the individual case of essential hypertension when a malignant phase of the disease will ensue. It occurs most frequently in the young. This malignant aspect of the hypertensive state frequently first manifests itself clinically by the retinal lesions already described; hence the prognostic importance of repeated ophthalmoscopic examinations in every patient with essential hypertension.

A word about the use of the term retinitis is necessary. It should be limited to the transudative and hemorrhagic reaction in the retina that is associated with the retinal vessel sclerosis. To designate the changes in all the structures—papilla, retina and arterioles—as retinitis is misleading. In a strict sense the term retinitis itself is a loose one. The lesions are not inflammatory, as "itis" would imply, but degenerative. At one time we spoke in a vague manner of "myocarditis." Now we limit that term to the purely inflammatory lesions of the myocardium. It has also been suggested that it is inappropriate to refer to exudates in the retina since most of the white spots seen with the ophthalmoscope are not exudates but serous transudates and hyaline, colloid or lipoid deposits.[13]

CONCLUSIONS

1. Retinal changes must be correlated with the modern knowledge of heart disease.

2. The failure of the authors of ophthalmologic textbooks to realize that arteriosclerotic heart disease and essential hypertension are distinctly different entities has caused much confusion.

3. The blood pressure is elevated in essential hypertension but not in arteriosclerotic heart disease.

4. Retinal changes in essential hypertension are quite different from those of simple arteriosclerosis.

5. The terms albuminuric and nephritic retinitis are etiologically inaccurate and should be discarded.

6. When referring to a patient with essential hypertension, the term arteriolar sclerotic retinitis should be used in place of arteriosclerotic retinitis.

7. Retinal arteriolar sclerosis is usually progressive and, once developed, is permanent.

8. The persistence of retinal arteriolar sclerosis permits of a diagnosis of essential hypertension even if the blood pressure is normal.

9. Prognostically, changes in the fundus are of definite value.

BITEMPORAL PARACENTRAL SCOTOMA

REPORT OF A CASE *

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PHILADELPHIA

It was because of two particularly interesting features that I desired to present this case of bitemporal paracentral scotoma: (1) the disposition of the scotoma unassociated with disease of the pituitary body and (2) the unusual etiology.

REPORT OF CASE

History.—J. R. K., a man, aged 36, consulted me in regard to his eyes on Sept. 2, 1930. He stated that in September, 1929, while living in Manila, P. I., he had been given injections of serum against typhoid, dysentery and cholera—all on the same day. A few days later, he suffered from an attack of blurred vision followed by severe pain in the eyes. He immediately sought the services of an oculist, who told him that he had glaucoma. Since then he had been using a miotic (0.25 per cent eserine), 1 drop in each eye, every other night. His general physical condition was excellent.

Examination of the Eyes.—The pupils were unequal, the left being larger than the right; they both reacted sluggishly to light and in convergence. The rotations were full. The anterior chamber was shallow. On palpation, the tension was apparently normal. By Schiötz' tonometer, the tension of the right eye was found to be 20 mm. of mercury; that of the left eye, 19 mm.

With the ophthalmoscope, the media of the right eye was found to be clear; the disk was of good color; the margins were hazy; there was no cupping; the veins were dark, tortuous and distended. There was some perivasculitis of the disk. The macula was dark and somewhat granular. Just above the macula, toward the temporal side. were observed two small, yellowish patches of exudate, about the size of a blood vessel, with clearcut and well defined margins.

The left eye showed the same changes, except that there were no patches of exudate.

Under homatropine, refraction revealed a low compound hyperopic astigmatism, with a vision of 6/6+ in each eye.

The blind spots were not enlarged. A small paracentral scotoma was mapped out just temporal to the point of fixation in each eye. Fields taken at varying intervals for the next four months did not show a change in these findings. The form and color showed moderate concentric contraction.

Special Examinations.—On September 3, urinalysis showed: slight cloudiness, an acid reaction, no acetone, 2 per cent of urea, no bile pigment, a specific gravity of 1.025, no sugar, straw color, no albumin, no diacetic acid, sulphates or mucin, a faint trace of indican and a normal amount of phosphates.

Microscopic examination revealed: no casts, a normal number of leukocytes, amorphous urates, no mucus, a normal number of epithelial cells, no cylindroids, no pus cells, no crystalline, no blood and a considerable number of bacteria. A specimen showed the presence of a considerable number of bacteria.

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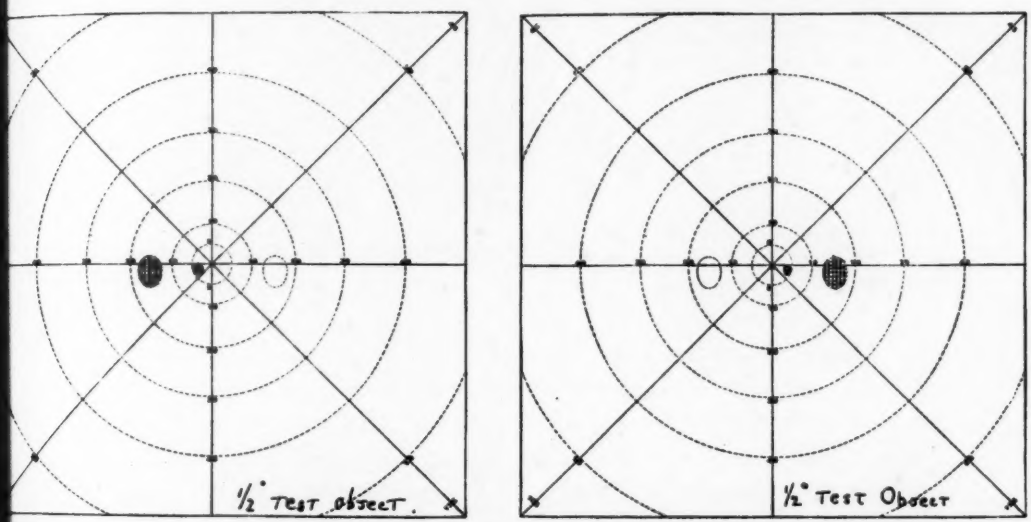
On September 5, the Wassermann and Kahn reactions of the blood were reported as negative.

On October 1, a blood count showed: hemoglobin, 96 per cent; index, 1; red cells, 4,590,000; white cells, 8,800. A differential count showed: polymorphonuclears, 63; small lymphocytes, 24; large lymphocytes, 8; transitionals, 1; monocytes, 1; basophils, 0; eosinophils, 3.

Dr. Fielding Lewis reported that the sinuses, nose and throat were normal.

Roentgenograms of the teeth showed two abscessed teeth; they were promptly removed.

Dr. Manges' report of a roentgen examination was as follows: "In the case of Mr. J. R. K. I beg to report that the bones of the skull are of average normal thickness and have normal vessel markings. There is a little thickening of the bone along all the suture lines. This is not an unusual thing but then it is not



Visual field of the left eye (on left side) and field of right eye (right side) taken on Sept. 3, 1930.

exactly normal. I think it indicates some little imbalance in the calcium metabolism perhaps, and, in the same way, there is a fairly large calcareous deposit in the region of the pineal gland. The sella turcica is definitely smaller than the average normal sella. There is some sclerosis around the left mastoid but there would have to be clinical evidence of trouble now in order to determine that it is an active mastoid process. The accessory sinuses are clear. There is now, I think, possibly very slight cloudiness of the right sphenoids—not marked at all."

A test of the spinal fluid gave negative results.

Dr. de Schweinitz [1] saw this case in consultation, and his report is as follows:

"I have just received a letter from Manges enclosing a copy of his report of the x-ray of the skull of Mr. J. R. K. The smallness of the pituitary fossa is interesting, but does not help much with respect to the diagnosis.

1. de Schweinitz, George E.: Concerning Certain Ocular Aspects of Pituitary Body Disorders, Mainly Exclusive of the Usual Central and Peripheral Hemianopic Field Defects, Tr. Ophth. Soc. U. Kingdom **43**:12, 1923.

"I cannot believe that these bitemporal small paracentral scotomas can be due to macular lesions, not only because the macula shows no definite signs of trouble, but on account of the disposition of the defects. Were these scotomas homonymous, they could easily be explained as an example of so-called median hemianopia.

"Now, as you know, Wilbrand and Saenger have described such small bitemporal, paracentral scotomas due to minute vascular lesions in the chiasm, usually syphilitic in character. I can think of no other explanation of the defect in Mr. K.'s field. I understand that the Wassermann tests of the blood were negative.

"That does not exclude syphilis, but I think a serologic test of the spinal fluid is in order. However, such minute lesions are not necessarily specific in character, and I should think might be caused by doses of various serums.

"I have seen such small paracentral scotomas gradually develop and bitemporal hemianopia appear in cases of pituitary dysfunction. But were such the case, this expansion of the scotoma should have begun long ago. Hence, I think minute vascular lesions in the chiasmal crossroads are the most likely diagnosis, and they doubtless are permanent."

COMMENT

There are four possibilities that might explain the scotomas: a tumor of the pituitary body, syphilis, a focal infection or injection of serums. Scotomas could occur with lesions of the pituitary body, but coincident with them changes are usually found in the peripheral fields. In the case reported, however, the peripheral fields constantly remained normal. The roentgenograms of the sella turcica were normal. Further, there was no increase in the size of the scotomas.

Traquair [2] reported the possibility of a scotoma in the cecocentral area due to a chiasmal retrobulbar neuritis in a case in which improvement occurred after the use of tobacco was stopped. In my case, there was no history of the use of tobacco. The possibility that sinusitis caused the condition was excluded by examination and by roentgenograms of all of the sinuses.

Syphilis could very well be the cause of scotomas. According to Wilbrand and Saenger,[3] if a small lesion acts on the posterior part of the chiasm from the floor of the recess, it will affect the crossed components of the papillomacular bundle. As a result, bitemporal, paracentral, absolute or relative scotomas are seen in the fields. They reported the case of a man, aged 20, with recently acquired syphilis, who showed bitemporal, paracentral scotomas, which were at first relative and later became absolute. Their explanation of this sparing of the macular region was based on the fact that the lesion did not involve all of the nerve fiber bundles of the crossed components of the papillomacular bundle. They also assumed that in the median

2. Traquair, H. M.: An Introduction to Clinical Perimetry, St. Louis, C. V. Mosby Company, 1927.

3. Wilbrand, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1915, vol. 6, p. 47.

sagittal plane of the chiasm, the crossing takes place in bundles and not as individual nerve fibers, for this is the most obvious explanation of the congruity of the defects in the fields.

They also reported the case of a man, aged 28, who had had a syphilitic infection ten years previously. The fields showed bitemporal, relative paracentral scotomas. The fundi were normal. The scotomas remained unchanged. A third case reported by them was that of a man, aged 28, with large, bitemporal, quadrantic, relative scotomas. The etiology was not mentioned. They quoted a case reported by Henschen in which bitemporal, paracentral scotomas disappeared after the application of mercurial inunctions.

In their opinion, the lesions are due to syphilis, which affects the chiasm and causes disturbances of nutrition. Signs of basal meningitis are not present. They also stated that central scotomas alone are not common in diseases of the pituitary body.

They explained the bitemporal, paracentral character of the scotomas as due to the vascular lesions in the crossed bundles of the chiasm.

Cushing and Walker [4] expressed the belief that the macular fibers, owing to their poor blood supply, are more susceptible to injury, though they occupy the center of the nerve at the chiasm.

According to Traquair, study of the chiasmal field defects proves that the crossed fibers intermingle before regrouping themselves in the tracts. Their mechanism of production is due to traction, pressure or toxins.

In the case here reported, as far as the findings show, chiasmal tumors and syphilis can be excluded.

In view of the findings it seems logical to suppose that the lesions were, undoubtedly, in the chiasm—minute vascular lesions of toxic origin arising from the rather drastic procedure of injections of serums against typhoid, dysentery and cholera given on the same day.

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4. Cushing, Harvey; and Walker, Clifford B.: Distortions of the Visual Field in Cases of Brain Tumor: Chiasmal Lesions with Especial Reference to Bitemporal Hemianopsia, Brain **37**:341, 1915.

LENS EXTRACT

ITS PREPARATION AND CLINICAL USE *

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In 1927, we [1] reported on the preparation of lens extract and its crystalline fractions. In more recent papers [2] we have gone into further detail concerning the preparation and purification of these fractions. No mention has been made of the use of lens extract, either as a therapeutic or as a diagnostic agent, although it has previously been used by several other workers.

Römer [3] used lens extract by mouth, and Davis [4] by subcutaneous injection, as a therapeutic agent in the treatment for cataract. In 1922, Verhoeff and Lemoine [5] described endophthalmitis phaco-anaphylactica, and announced the indications for treatment with lens protein for this specific condition. Their conclusions were later sharply challenged by Rotth,[6] and these objections were later replied to by Verhoeff. Berens and his co-workers [7] reported observations on several patients with hypersensitivity to lens extract who were treated by injections of such extract

* Submitted for publication, May 5, 1931.

* From the Wilmer Ophthalmological Institute, Johns Hopkins University.

1. Woods, A. C., and Burky, E. L.: Lens Protein and Its Fractions: Preparation, and Immunologic and Chemical Properties, J. A. M. A. **89**:102 (July 9) 1927.

2. Burky, E. L., and Woods, A. C.: Lens Protein: New Methods for the Preparation of Beta Crystallin, Arch. Ophth. **57**:41 (Jan.) 1928; Lens Protein: The Isolation of a Third (Gamma) Crystallin, ibid. **57**:464 (Sept.) 1928.

3. Römer, P.: Spezifische Therapie des beginnenden Altersstares, Ber. ü. d. Versamml. d. ophth. Gesellsch. **35**:195, 1908.

4. Davis, A. E.: Serum and Lens-Antigen Extract Treatment for the Prevention and Cure of Cataract, Tr. Am. Ophth. Soc. **22**:112, 1924.

5. Verhoeff, F. H., and Lemoine, A. N.: Endophthalmitis Phacoanaphylactica, Internat. Cong. Ophth., Washington, D. C., 1922, p. 234.

6. Rotth, A.: On the Question of Phacoanaphylactic Endophthalmitis, Arch. Ophth. **55**:103, 1926.

7. Berens, C., and others: Studies in Immunology in Relation to Ophthalmology: Outline of Diagnosis and Biologic Therapy, with Special Reference to Lens Antigen in Diagnosis and Treatment and Serum Therapy, Atlantic M. J. **30**:547, 1927.

both as a prophylactic before operation and as a therapeutic agent in combating postoperative inflammation after extraction of the cataract. These attempts at therapeusis with lens extract have not been based on the determination of a specific allergic state, except in the specific cases reported by Verhoeff and by Berens and his associates.

Lemoine and MacDonald [8] tested intradermally with lens extract 168 unselected cases of postoperative and traumatic injuries to the lens. Fourteen patients, or 8.6 per cent, gave positive reactions to the tests, and in all a fair amount of free cortical matter remained; intra-ocular inflammation developed in seven. These authors considered the intra-ocular inflammation to be allergic, owing to the absorption of lens protein in the specifically hypersensitive person. Gifford [9] tested 86 cases of cataract with lens protein preceding operation, and he found positive reactions in 12.6 per cent. Six of the patients giving positive reactions to the cutaneous tests preceding operation had stormy postoperative courses. However, eight patients with negative reactions to the tests had similar postoperative inflammation, and Gifford believed that postoperative reactions were due to an inherent toxicity of the lens matter left in the eye at operation, rather than to allergy. Experiments on animals confirmed the belief that the lens protein might be toxic, and greater toxicity was found in cataractous lenses than in normal lenses. Gifford believed, therefore, that while endophthalmitis phacoanaphylactica was a concrete entity, most of the intra-ocular inflammations that followed the retention of soft lens matter were due to inherent toxicity of the lens matter and to secondary protein-split products rather than to allergy. Courtney,[10] using a commercial preparation, tested twenty-six cataractous patients with intradermal injections of lens protein before operation. All of the patients gave negative reactions to the tests. Twelve were tested after operation, and five gave positive reactions to the tests. There appeared to be no relationship between the amount of lens cortex at operation and the development of sensitivity; four of the patients who gave positive reactions to the cutaneous tests after operation had apparently normal postoperative courses. Two of the patients, however, received a desensitizing course of injections of lens extract. The fifth patient had a typical endophthalmitis phacoanaphylactica.

Over a period of three years we have made tests with lens extract on the skin of normal persons and of patients with cataract. Patients

8. Lemoine, A. N., and MacDonald, A. E.: Observation on Phacoanaphylactic Endophthalmitis, Arch. Ophth. **53**:101 (March) 1924.

9. Gifford, S. R.: Allergic and Toxic Properties of Lens Protein, J. A. M. A. **85**:351 (Aug. 1) 1925.

10. Courtney, R. H.: The Use of Lens Protein Tests Before Cataract Operations, Am. J. Ophth. **12**:20, 1929.

with a demonstrated hypersensitivity to lens extract have been treated by subcutaneous injections of lens for certain specific conditions to desensitize either before or after operation or in the treatment for endophthalmitis phaco-anaphylactica. Since our first report our methods for the preparation of lens extract have been modified and improved, and a miscellany of information has been collected concerning its preparation and use. It is the purpose in this report, therefore, to present a brief summary of our present knowledge of the preparation and the use of lens extract.

PREPARATION

Beef eyes (150) are obtained from a slaughter house on the same day the cattle are killed. As soon as possible after the butchering, the eyes are opened by making an incision in the cornea at the limbus with a sharp pointed scissors. The incision is carried about halfway around the cornea. The aqueous escapes, and the encapsulated lens pushes into the opening. The capsule is punctured, and the lens escapes on the application of slight pressure. This method keeps the lens as free from aqueous as is possible.

The 150 lens are placed in a liter bottle containing broken glass, and the bottle is filled with hundredth-normal ammonium hydroxide containing 0.5 per cent, cresol, U. S. P. The mixture is shaken in a mechanical shaker for eighteen hours. After shaking, sufficient of the same solvent is added so that there is 20 cc. for each lens. The resulting emulsion is placed in the refrigerator for one week. At the end of this time, the emulsion is twice put through a Sharples steam driven centrifuge running 40,000 revolutions per minute. It is then filtered through a Berkefeld V filter for sterilization. To determine the strength of the finished product, samples of 5 cc. each are dehydrated at 104 C. in weighed crucibles. From the weights so obtained, the percentage strength is determined. After the strength has been determined it is diluted by adding sterile water containing 0.5 per cent cresol, U. S. P., and enough 8.5 per cent sterile sodium chloride to make the final concentration 1 per cent lens extract in 0.85 sodium chloride solution. Cultures to check its sterility are made.

In our earlier reports various methods were given for the clearing of the solution, and none was entirely satisfactory. Filtration through paper or Berkefeld filters is unsatisfactory because the lens fibers and insoluble residue soon clog the pores of the filter and establish an ultrafilter which lets through a clear but comparatively weak solution. Ordinary centrifugation does not remove all the insoluble material, and in addition it is a slow and laborious procedure. Centrifugation with a Sharples machine is the most satisfactory, and a clear solution is obtained, the total solid content of which is about twice that obtained by the earlier methods.

Lens extracts must be prepared with much attention to bacteriologic cleanliness and in the presence of a germicide, such as cresol, U. S. P. Lens substance is an excellent bacteriologic culture medium and is readily decomposed by bacterial action. Early in this study, the subjects and animals tested intradermally with lens extracts prepared without a preservative gave markedly positive reactions, although the preparations were sterile when used. When retested with more carefully prepared and preserved extracts, the same subjects and animals gave no reaction. Apparently the early positive reactions described were due to split-protein toxins produced by bacterial action.

ALLERGY OF THE SKIN

The test is performed by the intradermal injection of 0.1 cc. of a 1 per cent solution of lens extract. As a control, 0.1 cc. of 0.85 per cent solution of sodium chloride containing 0.5 per cent cresol, U. S. P., is injected. If the reaction is negative, the subject is considered to be insensitive to lens extract. However, if the reaction is positive, further tests are done. To determine quantitatively the reaction of the hypersensitive subject, higher dilutions of lens extract are injected in amounts of 0.1 cc. Considering the 1 per cent solution as 1 : 100, dilutions of 1 : 1,000, 1 : 10,000 and 1 : 100,000 in 0.85 per cent solution of sodium chloride containing 0.5 per cent cresol, U. S. P., are used. The tests are made on two occasions, because of the low incidence of positive reactions.

Certain subjects are hypersensitive to bovine protein. Traces of beef aqueous present in the lens extract may cause a false positive reaction in bovine protein-sensitive subjects. As a control, therefore, beef serum in a dilution of 1 : 1,000, containing 0.5 per cent cresol, U. S. P., and sterilized by Berkefeld filtration, is injected intradermally. Beef serum is used because it has the same antigenic properties as aqueous humor and is more easily obtained. Should this control be positive, the sensitivity of the lens can be determined by the use of an extract of pig or rabbit lens with similar controls of pig or rabbit serum. One striking example of this type of sensitivity was seen in a patient who gave a positive reaction to a commercial preparation of lens extract and to preparations of bovine serum and aqueous, but who gave a negative reaction to our preparation of both bovine and pig lens extract.

There are two types of positive reactions. The first type is similar to that seen in food or in pollen allergy. The reaction is characterized by a wheal, with or without erythema, which appears in about fifteen minutes and disappears after a few hours. It is especially important to control this type of reaction with beef serum. This type of reaction is most often seen in persons with diseases of the lens, without evidence of acute allergic reactions in the eye. The second type of reaction is the delayed or tuberculin type, which appears after twelve hours and reaches its peak in about twenty-four hours. It appears as a wheal with erythema, the wheal usually being as large as the area of erythema. If the wheal is more than 3 cm., the patient usually complains of pain in the swollen area. This type of reaction is most commonly associated with endophthalmitis phaco-anaphylactica.

RESULTS OF TESTS ON THE SKIN AND TREATMENT

During the course of the study, the skins of seventy-five or more normal persons were tested with lens extracts. Of this group, not one showed a positive reaction. Sixty-four patients with cataract were

tested. Of this number, eleven, or 17 per cent, showed positive reactions. With the exception of the reaction in one subject, all of the positive reactions occurred in persons over 60 years of age. The cases of the eleven patients giving positive reactions are summarized as follows:

CASES 1, 2 and 3.—The patients in these three cases gave positive reactions preceding operation. At operation, all of the lens substance was removed. Sequelae did not appear.

CASE 4.—This subject gave a positive reaction preceding operation. Two months after operation, intra-ocular inflammation developed. The patient reported on the condition, but did not return for further study.

CASES 5 and 6.—These two subjects gave positive reactions preceding operation. The picture was complicated by sensitivity to uveal pigment. In one case of postoperative inflammation uveal pigment was used with definite success.

CASE 7.—This subject gave a positive reaction and was given lens extract by subcutaneous injection preceding operation. There was a normal postoperative course.

CASE 8.—This subject gave a negative reaction preceding operation. Violent postoperative uveitis occurred. Sensitivity to lens extract and uveal pigment developed after operation. The patient received a long course of subcutaneous injections of both preparations, and the uveitis cleared. Two years later, before extraction of the cataract in the second eye, the patient was again treated with both lens and uveal pigment. There was a successful postoperative course. Six months later intra-ocular inflammation recurred in the first eye. The patient was again treated with lens extract and uveal pigment, and the inflammation cleared up.

CASE 9.—This subject was not tested preceding operation. There was postoperative inflammation. The cutaneous test gave a markedly positive reaction. Treatment was not given. The inflammation became chronic.

CASES 10 and 11.—These two subjects gave doubtful positive reactions to the tests preceding operation. Some lens substance was left behind at operation. The reactions of the skin became definitely positive synchronous with the development of endophthalmitis phaco-anaphylactica. The inflammation subsided with lens therapy.

Summarizing, seven of the eleven patients showed hypersensitivity to lens extract before operation. Three of these, in whom all of the lens substance was removed, had uneventful postoperative courses, while three others, in whom some of the lens cortex was left, had postoperative intra-ocular inflammation. The remaining patient was desensitized before operation, and had an uneventful postoperative course. Two subjects gave doubtful reactions preceding operation. Soft cortex was left at operation, and endophthalmitis phaco-anaphylactica developed in both patients. One subject gave a negative reaction before operation, but after operation hypersensitivity to lens and pigment developed synchronous with intra-ocular inflammation. The last patient was not tested before operation, but when postoperative inflammation developed, he was found to be definitely hypersensitive to lens matter.

Because of the small number of cases studied and the small group found positive, definite conclusions cannot be drawn. However, positive reactions of the skin were found only in subjects with cataract, in those with endophthalmitis phaco-anaphylactica or in those with inflammation following extraction of the cataract.

Our studies indicate that Gifford is correct in saying that lens protein or extract, as ordinarily prepared, may be toxic, probably owing to the formation of split-protein products. If carefully prepared, however, and if bacterial growth is prevented from the first stage of preparation, such lens extract has no toxic properties and appears entirely inert in normal persons. Certainly, no positive reaction of the skin to lens protein should be regarded as an indication of specific hypersensitivity, unless it is known that the lens protein used is inert in normal persons and that the reaction of the skin is not due to hypersensitivity to bovine protein. Our studies have not shown any inherent toxicity in lens protein, and we have seen nothing to indicate that postoperative inflammation may be due to such inherent toxicity of retained lens matter. We cannot agree with Gifford in this respect.

Several patients with postoperative uveitis and endophthalmitis phaco-anaphylactica were treated by subcutaneous injections of increasing doses of 2 per cent lens extract, the maximum dose being 2 cc. Such therapy appeared to be of definite value. No patient has been treated with lens extract for the cure or prevention of incipient cataracts. No comment, therefore, can be made on this treatment. We have, however, examined the blood serum of a patient so treated. He had received thirty daily subcutaneous doses of 10 cc. of 2 per cent solution of lens extract. His serum was collected on the ninth day following the last injection. No traces of precipitins or complement-fixing antibodies were found. For comparison, proportionate amounts per kilogram of body weight produce large amounts of antibody in rabbits. So far as could be determined, the therapy did not have any effect on the cataracts.

SUMMARY

The method of preparation of lens extract for use in tests on the skin has been outlined.

Negative reactions of the skin have been observed in seventy-five normal persons tested.

Positive reactions have been observed in eleven patients with cataracts, or 17 per cent. The majority of the positive reactions have been associated with postoperative inflammation after extraction of the cataract (in persons over 60 years of age).

Lens extract as ordinarily prepared may develop toxic properties, but when properly prepared it appears devoid of any inherent toxicity.

GUNSHOT WOUND OF THE OPTIC NERVE

REPORT OF A CASE *

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AND
PETER C. KRONFELD, M.D.
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Many cases of orbital injuries from gunshot have been reported. The effects of such wounds on the globe and its surroundings are well known. In most of the cases a bullet entered through the temple and lodged in the homolateral or contralateral orbit. Cases in which the entrance of the bullet was approximately the same, but the course of which was intracranial, are apparently infrequent, as we were unable to find an analogy to the following case.

REPORT OF CASE

A boy, aged 7, was brought to the hospital two days after receiving a gunshot wound of the head, inflicted by his father, who had suddenly become insane. The entrance of the bullet was 3 cm. in front of, and 5 cm. above, the right external auditory meatus.

The boy was inclined to drop off to sleep, but could be aroused easily. He answered questions and cooperated well. There was a slow but constant escape of blood-tinged cerebrospinal fluid from the left nostril. There was definite stiffness of the neck; Kernig's and Brudzinski's signs were present. There was analgesia of the right side of the face, especially about the right eye. The neurologic examination otherwise gave negative results.

The right eye was closed, and the patient could not elevate the upper lid even with extreme effort. With the left eye in primary position, the right eye was slightly divergent; the patient could not adduct, elevate or depress it, but he could abduct it to the normal extent. The eyeball did not rotate when he attempted to look down from an extreme lateral position. The right pupil was a little wider than the left; it did not react to light; however, it did react when light was thrown into the left eye. The right disk was hyperemic and slightly elevated and was bordered by numerous hemorrhages. The retinal veins were dilated, and the arteries slightly contracted. There was no perception of light in the right eye. The left eye was entirely normal.

The roentgenologist reported that the trajectory of the bullet could be traced along the entrance into the skull to its final lodging because of small fragments of lead that were torn off and deposited along the tract of the bullet. The bullet entered the skull on the right side just below the junction of the greater wing of the sphenoid with the temporal bone. The entrance wound to the skull was clean and sharp with practically no splintering of the inner or outer tables of the

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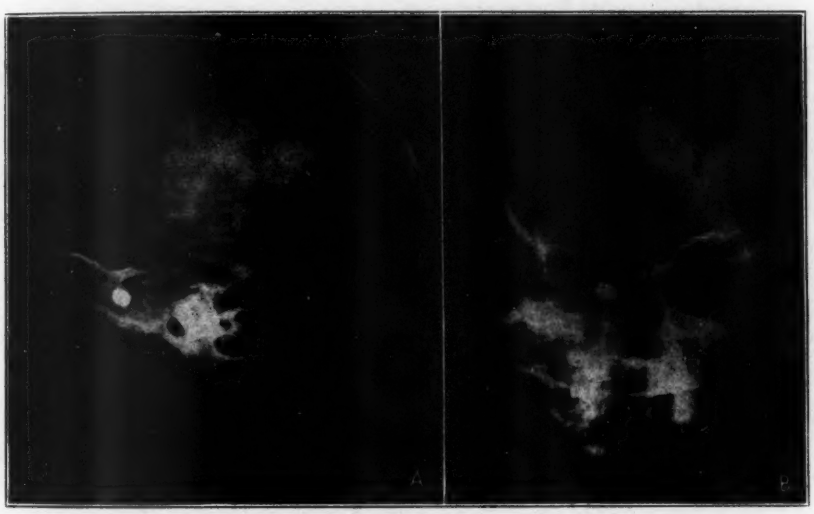
* From the Eye Clinic of the University of Chicago, Dr. E. V. L. Brown, Director.

skull. The path of the bullet was anteriorly and on a horizontal level; the tip of the right anterior clinoid process was broken off. After hitting the right anterior clinoid process, the bullet was deflected downward and came to lie one-fourth inch (0.63 cm.) anterior to the left anterior clinoid process, and one-eighth inch (0.31 cm.) below it. The bullet was one-fourth inch above the left foramen rotundum and on a sagittal plane with it. The nose of the bullet was pointing to the left. The bullet measured 9 mm. in length and 5 mm. in diameter. No other changes were seen on roentgen examination.

A provisional diagnosis was made of traumatic lesion of the right optic, oculomotor, trochlear and ophthalmic division of the trigeminal nerves; in other words, all the structures that run through the right optic canal and the superior orbital fissure, excepting the main vessels, were affected. It could not be determined which nerves had been severed and which ones had merely been interfered with by the extradural hemorrhage. The retina of the right eye never showed signs of ischemia.



A, the lateral view, shows the missile lying to the left and below the left anterior clinoidal process. The right anterior clinoidal process is broken off. Fragments of lead lie along the path of the bullet. *B*, the anteroposterior view of the patient's skull as seen from behind, shows the missile lodged behind the left orbit, on a level with its floor.

The patient's general condition improved, so that at the end of two weeks the rhinorrhea had ceased and all pathologic changes had disappeared except those of the right eye, which remained unchanged. The patient was discharged from the hospital to the outpatient department four weeks after the injury. In the fifth week the ability to elevate the right upper lid and to move the right eyeball began to return gradually. At the end of the fifteenth week the ptosis of the right eye had entirely disappeared, and the patient had full control over the lids. The eyes were parallel; the movements of the right eye were normal and followed those of the left accurately. The sensations on the two sides of the face and of the eyelids were equal. The left corneal reflex was a trifle quicker than the right, probably because the latter's reaction was the result of the visual plus the

sensory reflex. The right optic disk showed complete primary atrophy. The hemorrhage about the disk had all been absorbed. There was no perception of light from any part of the right retina; neither did the right pupil react to light thrown on any part of the right retina. The vision in the left eye was 1.5, and the left peripheral fields were normal for small white and red targets. The left fundus remained normal. This remarkable recovery of the mobility of the eye suggested that the lesion of the oculomotor, trochlear and ophthalmic division of the trigeminal nerves had been produced by extradural hemorrhage, which when absorbed allowed the nerves to function again. The right optic nerve was the only structure that had been severed. The patient has been under observation for twelve months and has not shown any symptoms of cerebral irritation from the bullet, which is still lodged near the tip of the temporal lobe.

COMMENT

Aside from the rarity of this type of injury, the case is unique in that recovery was almost complete. Even the fissure in the cribriform plate, which had to be assumed from the presence of rhinorrhea, became closed. The right optic nerve was the only structure permanently damaged, and its function was totally abolished. The lesion was apparently indirect, being due to the broken off tip of the right anterior clinoid process and not the bullet itself. The injury must have been limited to a rather small area in the intracranial course of the right optic nerve, since no gross damage was done to the ophthalmic artery or to those crossed fibers from the left eye which form the anterior loop of Wilbrand in the contralateral nerve. After fracturing the right anterior clinoid process, the energy of the missile had been spent so that it followed the line of least resistance without doing further damage. It apparently slid along the planum sphenoidale over the upper and lateral surface of the left anterior clinoid process and dropped downward to its final resting place, where it lodged in soft tissues.

The nerves and vessels that pass through the right upper orbital fissure were only temporarily affected. There the pathologic process most likely was an extradural hemorrhage, which by an increased local pressure abolished the function of the nerves. The pupillary fibers of the right oculomotor escaped because of their central location within the trunk of the nerve. The increased pressure on the ophthalmic vein caused the appearance of retinal hemorrhages. A hematoma of the sheaths of the optic nerve might have been present as a result of the fracture of the bone. The recovery of the function of the right oculomotor nerve took place after the boy left the hospital, which was four weeks after the accident; therefore, we do not know whether the levator or the rectus internus muscle recovered first.

The final outcome being unilateral blindness, the patient represented a pure picture of amaurotic fixation of the right pupil. If light fell into both eyes diffusely or from in front, the right pupil was always

slightly wider than the left. On lateral illumination, the anisocoria became more marked when the nasal half of the left eye was stimulated than when the temporal half was stimulated. These observations were made on several occasions and were verified by several observers.

Only those schemes of the pupillary pathway that assume a supra-chiasmal (second) crossing of more than 50 per cent of the fibers coming from one optic tract can explain the variations of the anisocoria that occurred on oblique illumination. Therefore, this case of amaurotic fixation supports the theories of Bach, Behr and Marques and speaks against the schemes of Levinsohn, Liepmann-Bumke and Groethussen.

THE DEVELOPMENT OF THE LENS

ITS SIGNIFICANCE IN THE INTERPRETATION OF LENTICULAR ABNORMALITIES *

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DEVELOPMENT OF THE LENS

The lens is peculiarly susceptible to variations from the normal because: first, in its development it must pass through a complicated cycle of changes wherein at any point its growth may become aberrant; second, it is surrounded by a number of equally mutable tissues which through their developmental defects may cause secondary deviations in the lens, and third, the delicacy of the lens offers comparatively little resistance to trauma and disease and predisposes to the formation of opacities. Many of these abnormalities are important from the standpoint of visual acuity, while others are relatively insignificant. All, however, are given added interest, and their clinical meaning is clarified when they are interpreted as phenomena occurring at some stage of the developmental history.

Human material suitable for study is scarce; even in animals it was difficult to obtain a complete series in any one species. I have been forced, therefore, to select sections from a number of series of different animals to illustrate the successive stages of growth. The eyes of all mammalian vertebrates are practically the same in their embryologic development, so that these illustrations may be accepted as representative of the corresponding periods in the human eye. Of course, the same stage in two different animals does not occur at the same age or length of the embryos; consequently, I have disregarded the question of length in labeling the photographs.

The anlage of the eye is definitive even before the closure of the neural tube as a lateral evagination of the neural plate. It is long axially, but soon becomes rounded.

The first chapter in the history of the lens may be visualized from figure 1. The neural tube has already closed along its entire length, and its head is divided by two constrictions into the prosencephalon, mesencephalon and rhombencephalon. The prosencephalon extends ventrad at right angles to the rest of the brain, and it is in this forebrain that the optic pits, now projecting as hollow round protuberances, called

* Submitted for publication, March 28, 1931.
* From the Knapp Memorial Eye Hospital.

optic vesicles, are located. The optic vesicles extend as far as the surface ectoderm, and lie in direct apposition with it. This adhesion is incomplete where the paraxial mesoderm, which lies along the central nervous system, intervenes along the sides and stalk of the vesicle. The paraxial mesoderm allows no such direct contact between the brain and surface ectoderm elsewhere. The optic vesicles point slightly caudad and ventrad. In microscopic preparations there is invariably a fine space between the surface ectoderm and the vesicles, and this space is bridged by fine protoplasmic processes extending between the adjacent sheets of cells and intimately

Fig. 1.—Optic vesicle in contact with the surface ectoderm. *a* indicates the amnion; *v*, the optic vesicle; *e*, the ectoderm, and *m*, the mesoderm.

connecting the two. No mesodermal cells are present in the interstices. (Bach and Seefelder, however, are of the opinion that in man, even before the closure of the medullary tube, the mesoderm lies between the optic bud and the ectoderm.) The cells of both the vesicle and the surface ectoderm are cuboidal and lie in single layers.

The cells of the surface ectoderm soon begin to differentiate. Thickening results when the cuboidal form is lengthened to a columnar shape. Mitotic figures and crowding of the nuclei indicate a rapid proliferation, but the cells maintain their arrangement in single layers, although at first this is not apparent. The nuclei are situated at different levels in the cells, and so impart the impression of several layers, but this is belied by a minute examination of the sections with the binocular

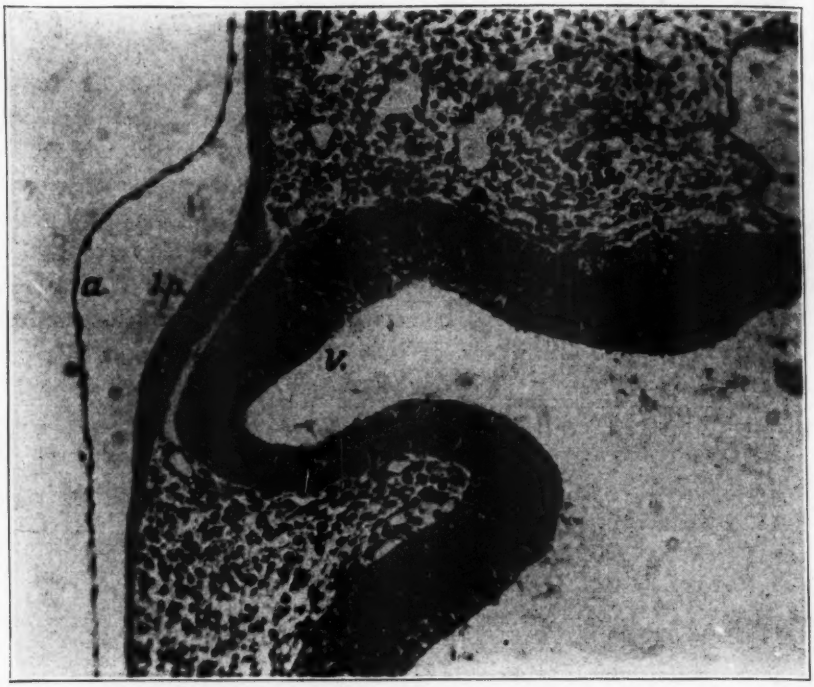

Fig. 2.—The lens plate. The amnion is seen covering the embryo. *a* indicates the amnion; *v*, the optic vesicle, and *l.p.*, the lens plate.

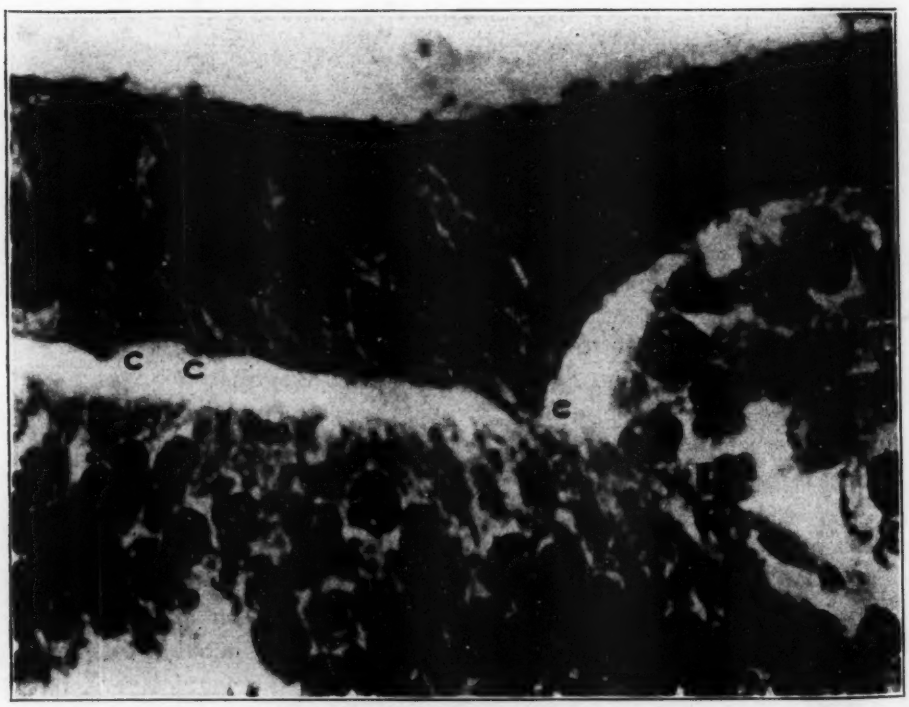

Fig. 3.—The lens plate, showing the arrangement of the nuclei. The bases of the conelike adhesions, but not the fibrils, may be seen (*c*).

microscope. The mitotic figures are mostly near the free or external boundary of the surface ectoderm. The protoplasmic bridges are even more pronounced and are cone-shaped. It is these processes that von Lenhossek claimed are the source of the vitreous body. They have a tendency to split in two at their tips. Still no mesoderm is found in the space, although an occasional proliferating cell may project past its neighbors and lead one to believe that a third cellular element is present. The thickened group of cells of the surface ectoderm is known as the lens plate; it presents a gentle depression in its lower part (in non-mammalian vertebrates in the center), the fovea lentis. Soon the fovea lentis deepens to form the lens pit. The lens pit contains in its hollow amniotic fluid and some free cells.

Fig. 4.—The lens pit. Amniotic coagulum may be seen in the hollow. *v* indicates the optic vesicle. The space between the anterior and posterior layers is exaggerated through shrinkage of the tissues.

The optic vesicles have invaginated correspondingly to receive the ingrowing lens. Mesodermal cells may possibly now be seen between the lens and optic vesicle, but they are rare. They appear as the forerunners of vascular channels. The lens continues to deepen, and the edges of its mouth to approximate, so that a lens vesicle is formed, hanging from the surface ectoderm by the lens of the stalk.

A still closer approximation of the lips of the vesicle, a pinching in of its stalk, and the result is a hollow sphere lying free below the surface of the ectoderm and containing in its lumen the imprisoned cells and amorphous material already noted (fig. 7). These cells have no clinical import, but they are of interest for theoretical speculation. Ida Mann considered them epitrichial, believing that the differentiation of the sur-

face ectoderm to form the epitrichial or squamous layer is more advanced in the lens pit than elsewhere. Bach and Seefelder believed these inclusion cells to originate from the wall of the lens, and in certain specimens demonstrated apparent connections. In these areas, also, mitotic figures are more evident, giving the impression of local proliferation. They also found a few similar cells breaking off toward the vitreous cavity. Some of these cells show signs of degeneration, and according to Bach and Seefelder they are extruded from the lens during the process of growth. The invading mesoderm is much more apparent now both externally

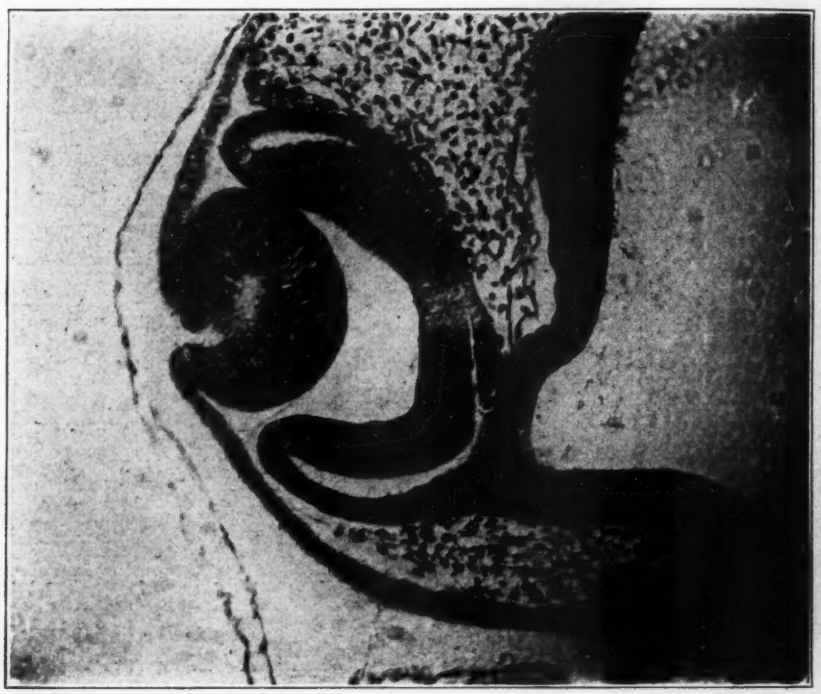

Fig. 5.—The lens, which is not quite closed, is about to be snared at its stalk.

and internally to the lens, and it carries with it newly formed endothelial channels containing the nucleated red blood cells of the fetus. The cells of the lens vesicle are unaltered, being columnar and single layered.

The cells of the posterior wall now elongate, their nuclei moving forward apace. The lens cavity is encroached on and rendered crescentic. The growing mass of lens fibers has been called from its appearance the "linsenpolster" or lens cushion. The central fibers form a straight line; the peripheral fibers, a concave line, with the concavity outward. The nuclei are found more forward, probably because the fibers grow faster in a backward direction. The nuclei of the central fibers stain less strongly.

A definite line of condensation may be seen as the site of the hyaline capsule of the lens. The capsule is thought by most writers to be a secretory product of the lenticular cells, most likely of the anterior wall. Up to this time numerous cone-shaped fibrils have been seen extending in all directions from the lens, but with the laying down of the hyaline capsule it is difficult to find them, and then only in badly shrunken sections. The cells of the anterior wall of the lens do not elongate; they are thinnest in the center. Their nuclei become evenly placed. They play no further rôle in the development of the lens than

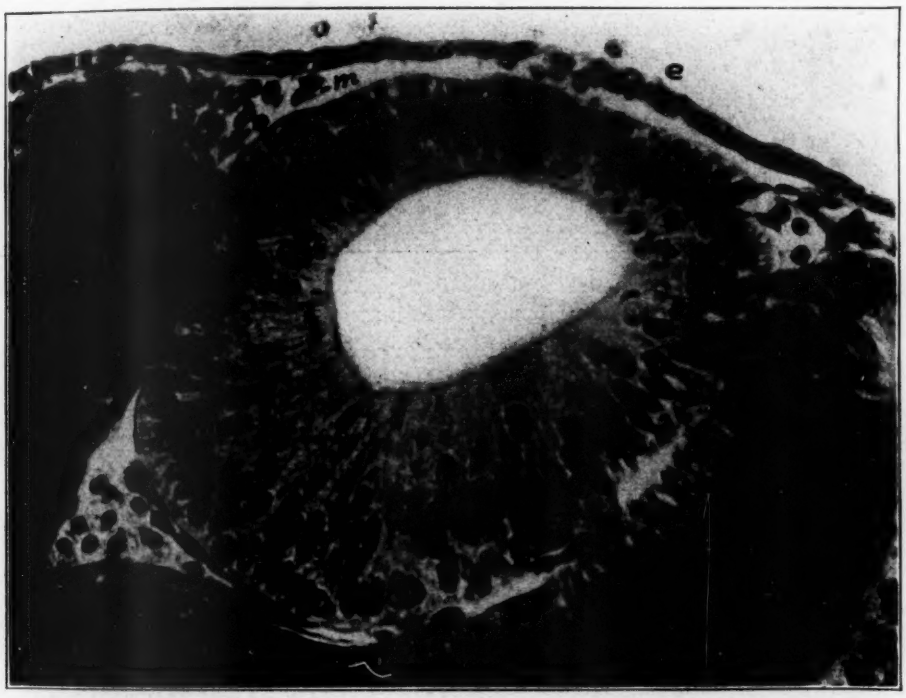

Fig. 6.—The lens is now a complete hollow sphere lying below the ectoderm. The cells of the posterior wall are beginning to elongate. *e* indicates the ectoderm and *m*, the mesoderm.

in the development of the epithelium anteriorly. The cells caught within the lumen of the lens disappear when the cavity becomes obliterated.

There is only a potential interval between the capsule-covered epithelium and the primary lens fibers. In shrunken sections, this space becomes apparent. The capsule of the lens is well shown in figure 8. In this specimen the posterior portion is the thickest, whereas in man the anterior half of the capsule is the better developed.

The amount of mesenchyme varies in different animals. Figure 8 shows a cross-section of the eye of a white mouse soon after the lens

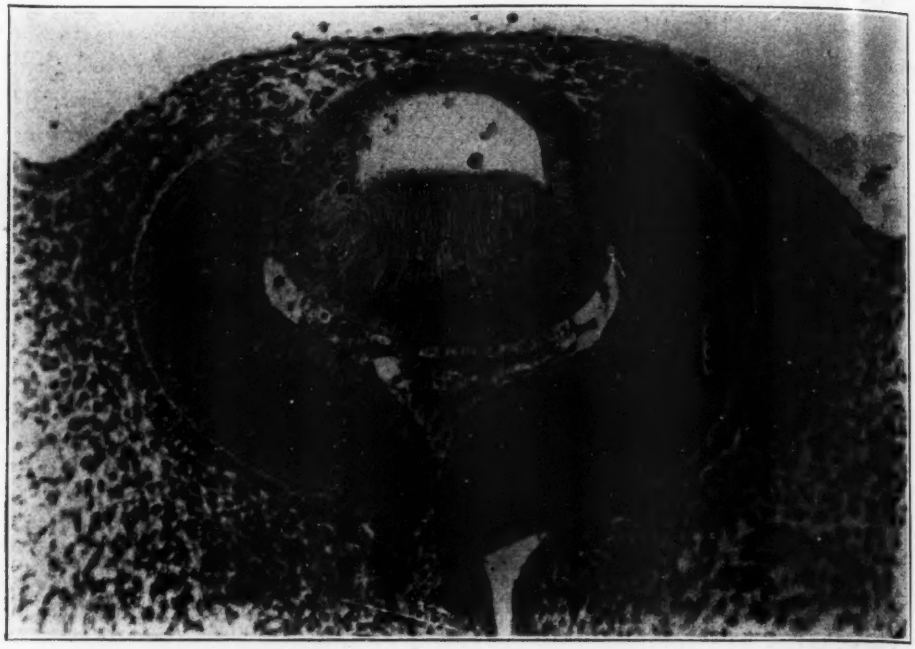

Fig. 7.—Further elongation of the posterior cells of the lens vesicle. Free cells may be seen within the lumen. The nucleated red cells are distinct. The mesoderm now lies between the lens vesicle and the ectoderm.

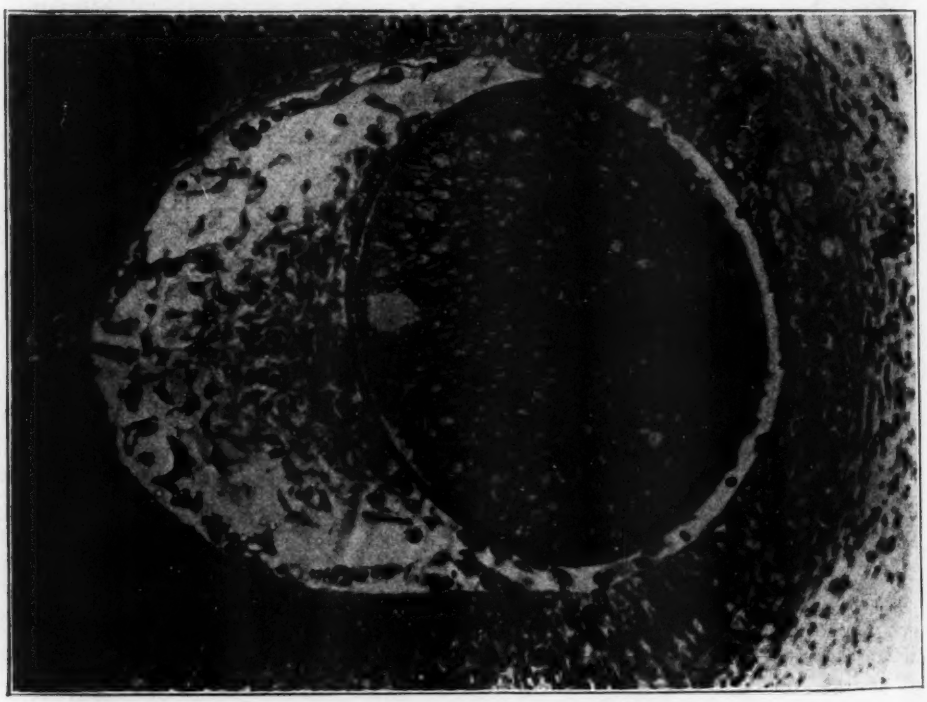

Fig. 8.—The hyaline capsule is well demonstrated. Behind the lens lies a cone of mesenchymal tissue. The tunica vasculosa lentis may be seen.

vesicle has become filled by the growing fibers. Directly behind the lens may be noted a conical mass of mesenchymal cells, the apex of which points toward the retina. The mesenchyme tends to collect at the posterior capsule, and at a later period it condenses to form a thick pad of cells which fits snugly against the back of the lens. This phenomenon seems not to have been described heretofore. It is possible that this formation is peculiar only to the mouse, but I believe it more likely that it has been overlooked in other species and in man because of its transitory nature. The cells are tightly packed; in cross-section the pad appears crescentic. The nuclei are arranged parallel to the circum-

Fig. 9.—The mesenchymal pad behind the lens. The arteria hyaloidea is seen entering the substance of the pad (*m.p.*). From the collection of Dr. Mihalik.

ference of the lens; they are distinct, oval, with a lightly staining protoplasm which contains a nucleolus and numerous small granules. Occasionally a cell may be seen from the pad extending into the vitreous. The pad covers about two thirds of the area of the posterior surface of the lens. The hyaloid artery reaches the center of the pad and there breaks into numerous branches which may be seen to course through the substance of the pad.

The mesenchymal pad is probably connected with the growth of the vitreous. It is present between the 10.5 and 15 mm. stages in the white mouse. The space between the lens and the retinal cup has become

greatly enlarged, and it is quite apparent that here the mesenchyme plays a major part in the building of the vitreous tissue. The mesenchymal cells soon disperse and become fewer in the growing vitreous space, but the pad behind the posterior capsule of the lens probably serves as a mesenchymal reservoir to allow the formation of the vitreous to keep pace with the rapidly expanding vitreous space. No trace of the pad can be seen in figure 10. Further investigation on this structure is needed.

Fig. 10.—The mesenchymal pad has disappeared. An artificial cleft between the epithelium of the lens and the fibers of the lens is produced by shrinkage of the section. The hyaloid artery is clearly shown. *t.v.r.* indicates the tunic vasculosa retinae, consisting of branches of the vasa hyaloidea propria applied against the retina. This is the source of the future permanent blood supply of the retina. This condition is not found in man; only in certain animals. This specimen is from a white mouse.

In the meantime the lens has been enlarging by proliferation of fibers from the equatorial zone. Here the nuclei are crowded together, but are dark and clear in contradistinction to those in the center, which are sparser and evidence the stigmas of degeneration and which are soon to disappear. The newly formed secondary fibers course forward and backward from the equator, but always each layer of fibers at its birth lies directly under the epithelium in front, and under the posterior

capsule behind. The newest ones lie superficial to the older ones and compress them as they multiply. The individual fibers are long; the nuclei lie at the zonular ends, while the other ends are spatulated to allow for more nearly perfect union at the suture lines.

The addition of the secondary fibers effects a change in the shape of the lens as a whole. At the beginning of their growth the lens is quite round, but soon the lateral diameter exceeds the anteroposterior,

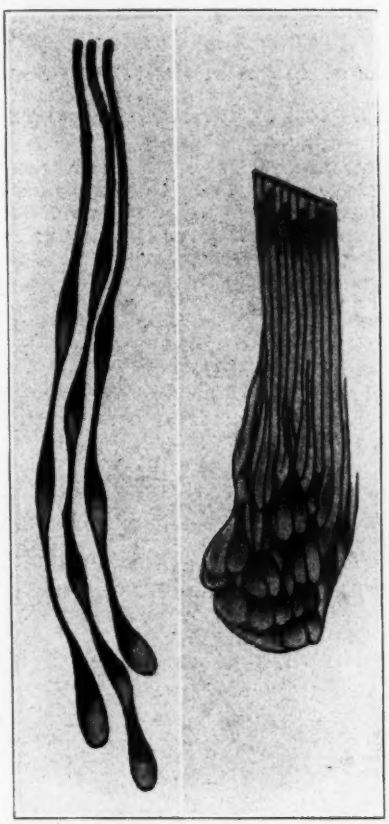

Fig. 11.—Lens fibers, showing spatulation of ends at the suture lines. (After Rabl.)

and the lens assumes a flattened appearance which becomes more accentuated after birth and even more so as the individual becomes older.

The development of the sutures is interesting. We have seen that the primary lens fibers need no suture line since they extend directly forward to the anterior wall of the vesicle. But the secondary fibers, to preserve the optical properties and shape of the lens, must lie regularly and unite with economy, an arrangement best attained by the suture lines. These lines of adhesion extend through the entire thickness of the lens at all times (except in the region of the primary

fibers of the optic vesicle). One is apt to think of the sutures as existing only in one or two planes because they appear so under a cursory examination with the slit-lamp, but this impression is due to the fact that the refractive index of the several layers of the lens is not uniform, and at these surfaces of discontinuity the suture markings are optically strongest.

To Vogt in particular is credit due for a classification of the zones of the lens, but the slit-lamp classification is not quite consistent with the embryologic conceptions. I propose a slightly modified classification based on the time of origin of the several zones:

Present at birth	1. A central clear interval. This corresponds to the primary fibers of the optic vesicle. There are no suture lines. Growth ceases at the end of the second month.
	2. The embryonic nuclei. These are separated by the central clear interval. The nuclei are formed during the first period of gestation. Their shape is planoconvex, with the flat surfaces facing each other. Growth begins at the second month and ceases at the eighth month.
	3. The fetal nucleus. This is formed during the latter period of gestation. Growth begins at the eighth month and continues to form during childhood till about the age of 10.
Formed after birth	4. The puerile nucleus. This represents the growth of the lens beginning at 10 years up to early adult life.
	5. The adult nucleus. This portion is formed during adult life and senility.

One must not confuse this division with the surgical classification of nucleus and cortex. One must not suppose also that all the zones and sutures are to be seen clearly in any one individual; the composite picture must be visualized from a number of lenses.

The shape of the sutures varies at each zone, and the shape at each zone varies considerably in different individuals, but in general the shape of the suture is as follows:

(*a*) At the anterior layer of the embryonic nucleus, it is that of an upright Y.

(*b*) At the posterior layer of the embryonic nucleus, it is that of an inverted Y (Λ)

(*c*) At the fetal nucleus, it is starlike.

(*d*) At the puerile nucleus, it is that of a combined upright and inverted Y (Y)

(*e*) At the adult nucleus, it is starlike, with many radii and bifurcations of the radii.

It is to be remembered that at birth the star-shaped sutures of the fetal nucleus lie just under the capsule, but the layers of the fetal nucleus at this time are so thin that the suture lines of the embryonic nuclei are much more prominent. The earliest vitreous is supposed to be formed by protoplasmic processes extending from the lens plate

and then from the surfaces of the optic vesicle and the concavity of the retinal cup. The quantity formed from the lens, however, cannot be great, for the laying down of the hyaline capsule precludes any further development. The vitreous fibrils adhere to the surface of the capsule and the blood vessels around the lens and form a line of condensation, the capsula perilenticularis fibrosa, which comes to include in its sheath the lens and contiguous very fine primary vitreous. Ultimately, the walls of Cloquet's canal become continuous with the capsula perilenticularis fibrosa. The subsequent denser and avascular vitreous is called the

Fig. 12.—A cone-shaped fibril extending from the lens pit to the retina. *l* indicates the lens; *r*, the retina, and *c*, the cone-shaped fibril.

secondary vitreous, and in its growth it lies against the primary portion, the two being separated by the capsula perilenticularis fibrosa. The capsula perilenticularis fibrosa is here analogous to the hyaloid "membrane" of the adult in back of the retrolental space. Miss Mann pointed out that the slit-lamp picture in the adult of the almost optically empty retrolental space in which a very fine fibrillar structure may be seen under high magnification, and the vague but recognizable hyaloid "membrane" in front of the firmer vitreous, may possibly have their counterparts in the fetal, thin, primary vitreous and the capsula perilenticularis fibrosa.

To understand the presence of certain vascular remnants connected with the lens, one must understand the circulation around the fetal lens. It has already been seen (fig. 7) that the mesoderm entering the eye through the fetal cleft carries with it a plexus of blood vessels, at first disorderly in their arrangement, but soon becoming definitive as the arteria hyaloidea and its branches. The hyaloid artery courses forward to the posterior capusule a little below and nasal to the center, and there, in the fibrillar network of the capsula perilenticularis fibrosa, breaks up into numerous divisions which ramify over the whole of the posterior surface of the lens and past the equator to constitute the tunica vascu-

Fig. 13.—Equatorial section through a human lens. The tunica vasculosa lentis is very prominent. The posterior suture of the embryonic nucleus is shown.

losa lentis, well seen in figure 13. Before reaching the lens the hyaloid artery gives off a series of arteries roughly divisible into an inner and outer set, the vasa hyaloidea propria. These ramify extensively through the vitreous and hyaloid canal, and anastamose with each other and with the vessels of the tunica vasculosa lentis. The latter send forth straight parallel branches, the lateral or capsulopupillary divisions of the tunica vasculosa lentis, across the equator of the lens and internal to the margins of the optic cup, to drain into the annular vessel. This annular vessel encircles the optic cup at its margin and receives blood also from the primitive choroidal network growing on the outside of the cup. As the capsulopupillary vessels pass forward to the annular

vessel they must curl over the lips of the optic cup, and here in places they form grooves in the margin of the cup. Normally these grooves disappear with the recession of the vessels, but occasionally they persist as atypical colobomas of the retinal part of the iris and ciliary body.

The mesoderm lying in front of the lens and separating it from the surface ectoderm divides into two layers, with a fine space, the anterior chamber, between them. The anterior layer is adherent to the surface ectoderm and is never vascularized, but the posterior layer, lying in contact with the front of the lens, becomes invaded by a number of short loops from the annular vessel. These loops, with their matrix of mesenchymal cells, form the pupillary membrane. The pupillary membrane appears like a perforated diaphragm across the open mouth

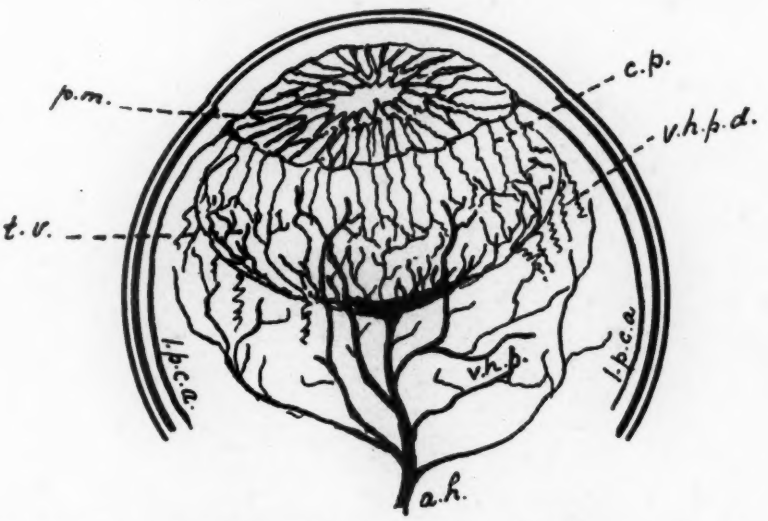

Fig. 14.—The early perilenticular circulation. *a.h.* indicates the arteria hyaloidea; *v.h.p.*, the vasa hyaloidea propria; *l.p.c.a.*, the long posterior ciliary artery; *p.m.*, the pupillary membrane; *t.v.*, the posterior vessels of the tunica vasculosa lentis; *c.p.*, the lateral or capsulopupillary vessels of the tunica vasculosa lentis in process of atrophy; *v.h.p.d.*, degenerating remnants of the vasa hyaloidea propria. The annular vessel into which the capsulopupillary vessels had emptied has disappeared. (After Versari, modified.)

of the optic cup. It can be seen as composed of a thin central portion and a thicker, more cellular peripheral portion.

In the meantime the edges of the optic cup have been extending forward as the ectodermal layers of the future iris, and in passing the equator of the lens have compressed and atrophied the capsulopupillary vessels, thus severing the vascular connections of the pupillary membrane with the intra-ocular blood supply. The annular vessel also disappears. The growing iris now lies under the peripheral portion of the pupillary

membrane, and carries with it arterial twigs from the long ciliary arteries, and it is from these vessels that the pupillary loops now receive their blood supply. These loops send out additional loops until three or four concentric sets or arcades span the pupil, all but closing it. None of these loops passes entirely across the pupil, but they return to the region from which they started. The first row of loops contains the thickest vessels; the central loops are finer. The central loops gradually become occluded and disappear, but the thicker peripheral row remains as the superficial layer of vessels of the iris, readily rendered conspicuous

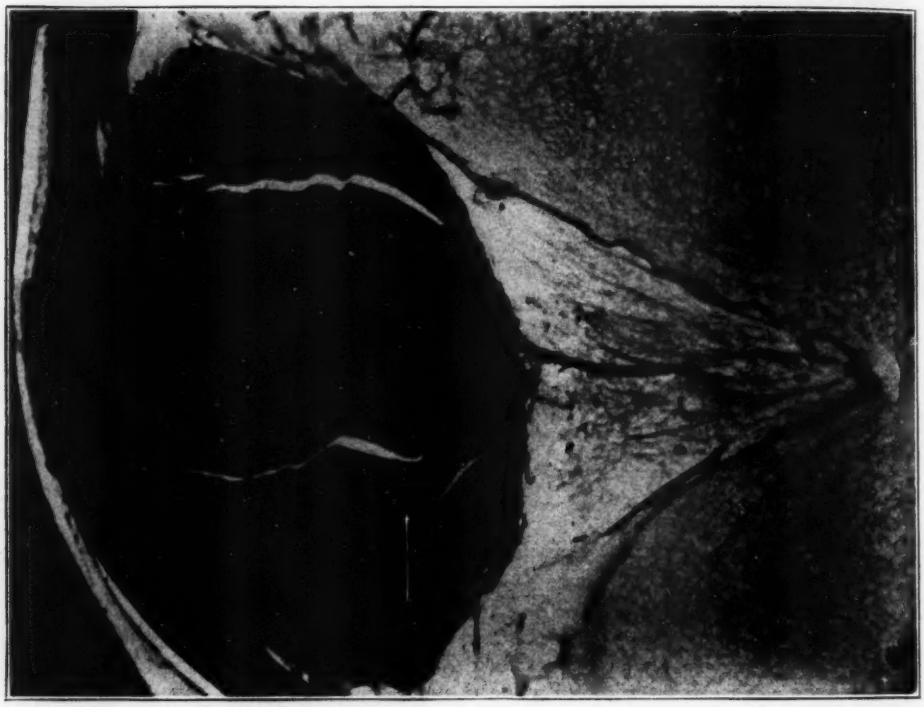

Fig. 15.—Cloquet's canal. Note the difference in texture between the primary and the secondary vitreous.

by an attack of acute iritis. The boundary between the peripheral and second rows of loops persists as the lesser circle of the iris. A cleft (Fuchs') forms between the lesser circle and the underlying true iris, thus separating the two layers for a variable distance.

The vasa hyaloidea propria have also been undergoing a process of atrophy, beginning at their origins from the main vessel by a constriction and subsequent severance. Their distal ends hang free from the posterior surface of the lens until they too atrophy. They curl into cork-screw shapes by virtue of the elasticity of their walls. The larger central vessels are the last to disappear; ranged along the parent hya-

loid artery, they form a funnel-like tree which lies embedded in a loose vitreous framework. This framework represents part of the primary vitreous. It may be distinguished from the later avascular secondary portion (formed during the atrophy of the vasa hyaloidea propria) by its central location, rarity, the presence of the hyaloid artery and its branches and a distinct line of condensation at its outer limits. This is the canal of Cloquet. The hyaloid artery shares the same fate as its branches, and during the last month of fetal life hangs pig-tail fashion in Cloquet's canal, adherent only to the posterior part of the capsule.

While the capsulopupillary vessels are in the process of atrophy, the ciliary processes have been forming by folds in the layers of the optic cup close to the margin. These folds elongate and approach the equator of the lens, and by the fourth month their tips are in temporary contact with the equator and adjacent posterior surface. Their points of contact produce little depressions in the contour of the lens. From the surface of the cells, strong fibrils arise in the valleys of the ciliary folds, and with the equator of the lens as their goal, cut their way through the intervening vitreous, to adhere to the zonular lamellae of the hyaline capsule.

SIGNIFICANCE OF THE DEVELOPMENT OF THE LENS IN THE INTERPRETATION OF LENTICULAR ABNORMALITIES

In the consideration of lenticular abnormalities one must distinguish between those that are (*a*) due to an arrest of development at a certain prenatal stage (these are obviously congenital); (*b*) those which represent embryonic structures normally fated to disappear, but which fail to follow their prescribed destiny (also congenital), and (*c*) those that are pathologic in the sense that they are unrelated to the embryologic cycle but are due to trauma, toxins or dystrophic influences (either congenital or postnatal).

(*a*) *Lenticular Defects Attributable to Arrests of Development.*—Aphakia: A number of observers have recorded the absence of a lens, but always in eyes that were also otherwise congenitally malformed (microphthalmic or cystic eyeballs). Aphakia, per se, if it existed, would be due to a failure to the lens vesicle to form.

Microphakia: This abnormality is due to cessation of the development of the lens at an early period of postnatal life. There may be an accompanying relaxed and stretched zonular ligament. The lens may at times prolapse into the anterior chamber, to return behind the iris on the assumption of recumbent posture. When the pupil is dilated, a considerable space may be seen between the border of the lens and the iris. The lens is usually well shaped but slightly more globular than usual, and the refractive error is one of myopia.

Coloboma of the Lens: This defect occurs when a sectoral portion of the lens fails to develop properly. The notch is usually downward, down and out, or down and in, and the defect corresponds to a weakened or underdeveloped sector in the suspensory ligament of the lens. At times there is an associated coloboma of the choroid or iris. This is not surprising since the coloboma of the lens occurs in the position of the fetal cleft. Usually, but not necessarily, the eye is myopic. Bowman advanced the theory that a feeble zonular ligament is the responsible factor. Heyl believed that an inadequate or arrested growth of part of the tunica vasculosa lentis caused a nutritional disturbance which resulted in the arrest of development of that part of the lens. Hess also laid the blame on the tunica vasculosa lentis but considered that the coloboma is caused by pressure exerted by some of the fetal vascular tissue which has failed to undergo regression and resorption at the usual time. Bock expressed the opinion that a finger of the ingrowing mesoderm interferes with proper development of the lens at one place. Bach attributed the coloboma to a disparity in size between the growing lens and the surrounding optic vesicle and the mesoderm. At the point of strongest contact the pressure induces a degeneration and absorption of the subcapsular fibers with the inevitable notch. The generally accepted theory is that lessened traction from weak or absent zonular fibers induces the notching. Clinical observations frequently confirm the absence of the zonular fibers. It is stated that the defective edge of the lens is more globular than the rest on account of the lack of stretching by the suspensory ligament.

Ectopia Lentis: Displacement of the lens is another sequel to a defective development of the zonular fibers. As the ciliary body grows away from the lens, the traction on the lens is unequally distributed. The lens is usually displaced upward, upward and inward, or upward and outward. The weak portion of the zonule is seldom or never above, consequently the displacement is seldom or never downward. A coloboma of the lens may exist at the same time.

Posterior Lenticonus: In most cases a gap is found in the posterior capsule; when no break is present, the posterior capsule is thinned out. Through the gap, lens fibers may project into the vitreous. Hess has examined the lenses of a number of animals which showed a nucleus not central, but lying close to the posterior capsule, either at the posterior pole or between the posterior pole and the equator. In some of these the condition of lenticonus was evident, being produced by the nucleus bulging backward, or by a stratum of lens fibers which had grown over the retrodisplaced nucleus. The posterior capsule was, as a rule, unbroken, but some specimens gave evidence of rupture during the fetal stage between the posterior pole and the equator. Through the aperture an irregular eminence of lens tissue projected into the vitreous.

Bach found three cases of posterior lenticonus in rabbits. Partial cataracts existed in all. The hyaloid artery remained as a thick band of connective tissue affixed to the posterior pole. The posterior capsules were thinned or ruptured, and the posterior surface of the lens was either conical or globular. Bach believed that the contraction and pull of the hyaloid artery was the basis of formation. There are, however, cases of posterior lenticonus in which the hyaloid artery cannot be demonstrated. It has also been suggested that the rupture of the posterior capsule is caused by a relative increase in the size of the lens, but no evidence can be presented to substantiate this.

Anterior Lenticonus: This anomaly is explained by a mishap at the time the lens vesicle is about to separate from the surface. For some reason the separation may be delayed, and the anterior portion of the lens becomes cone-shaped or, more rarely, globular. The conical part of the lens is more highly refractile than the periphery.

Anterior Polar Cataract and Pyramidal Cataract: These defects are caused at the time the lens vesicle is still open. A growth of fibers from the posterior wall forms an extrusion which remains as a projecting opacity on the front of the lens. The anterior capsule, being a later secretory product, covers the opacity. Another theory is that the amnion pushes itself into the open lens pit and for a while mechanically interferes with the closure.

Disk-Shaped Cataract: Treacher Collins has described a cataract, dumb-bell-shaped in cross-section, the center of which is composed of a stratified tissue representing multiplications of the epithelial layer. The explanation offered is that the cells of the posterior wall fail to elongate, but the cells of the equator proliferate as a rim of unnaturally formed lens fibers.

Congenital Zonular Cataract: The origin of this cataract is similar to that of the disk-shaped cataract; the fibers of the posterior wall do not elongate, but here the subsequent equatorial fibers cover the center of the lens. In their growth, however, on the irregular core, they themselves become irregular and opaque.

Congenital Morgagnian Cataract: In this type of cataract, the central fibers are more or less normally formed, but the equatorial fibers become dystrophic, degenerate and liquefy.

Axial Fusiform Cataract: This cataract consists of a slender spindle-shaped opaque area which extends through the anteroposterior thickness of the lens, the thick part of the spindle lying in the center. The attempt to explain this type of abnormality as the result of an adhesion of the nucleus to the anterior and posterior capsules fails to be fully convincing.

(*b*) *Abnormalities Due to a Persistence of Fetal Structures.*—Tags of Persistent Pupillary Membrane: This defect is caused by the

failure of some of the vascular arcades to atrophy. The tags originate always from the heavier loops in the circulus iridis minor. When the cleft of Fuchs is enlarged, the superficial layer of the iris is often undermined extensively and separated from the deeper iris tissue at the lesser circle. Bits of persistent pupillary membrane may be seen hanging from the iris in such cases.

Pigment on the Surface of the Lens: Pigment on the anterior surface of the lens is often associated with persistent pupillary loops, or it may exist alone. The pigment originates from chromatophores which have been misplaced on the front of the lens. The units of pigment are stellate in shape. Deposits of pigment on the lens following iritis have a tendency after a time also to become stellate, and unless other extenuating evidence is present it may be impossible in a given case to say whether such pigment is congenital or acquired.

Pigment on the posterior surface or the equator is rarer. These clumps denote the points of early contact between the lens and the ciliary body.

Posterior Polar Cataract: This type of cataract is usually interpreted as the result of the persistence of a portion of the tunica vasculosa lentis. Microscopic examination of the opacity, however, shows an amount of tissue which, to my mind, cannot readily be accounted for by the vascular network. I believe that the answer is to be found in the thick mesenchymal pad of cells behind the lens. A recent case reported by Leech bears a remarkable resemblance in section to the crescentic mesenchymal pad.

Persistence of the Hyaloid Artery: This anomaly is not very rare. It appears as a vessel extending from the disk to the posterior pole of the lens, sagging in its middle due to the action of gravity. It is sometimes associated with a posterior polar cataract. Its insertion into the lens is often nasal to the midline. The persistence of the distal end of the hyaloid artery is very common and may be seen with the slit-lamp in the majority of young children. It hangs down behind the lens as a curled tag in Cloquet's canal.

Remnants of the tunica vasculosa lentis are occasionally seen dependent from the back of the lens in eccentric locations. They are one of the causes of muscae volitantes.

The persistent hyaloid artery may at times be seen associated with the formation of fibrovascular tissue in the vitreous just behind the lens. In these cases the mesodermal cells have continued to proliferate beyond their usual time. The mass of cells is sometimes mistaken for a glioma.

The Canal of Cloquet: This canal often persists in the adult, but it drops below the optical axis of the eye and therefore is not readily

apparent in an eye that is in the upright position. The circular border of its mouth may be seen as an arcuate line, containing in its center the tag of the hyaloid artery. The nasal border is the more frequently seen, possibly because in the rotation of the orbital axis medially more tension is put on the lateral border of the canal mouth and it gives way there.

(*c*) *Pathologic Conditions.*—Under this head are included those opacities that are not brought on by developmental aberrations. They need not be listed individually. They are to be considered as degenerative processes following trauma, disease (local or general) or disturbed nutrition. For instance, the nuclear sclerosis seen in elderly people is due to compression of the central portion by the newer growth of cells and at the same time also to the poorer nutrition common to all the tissues in senility. The newer fibers of elderly people are not as homogeneous as those formed while young; even when the nucleus is clear one may see opacities in the cortex.

One may tell, by observing the depth at which an opacity is found, approximately in what time of life it was formed. Thus, the age of a sutural cataract may be determined by its depth. But this rule must be qualified with the statement that an opacity may form during middle age in an earlier stratum. To be exact, then, one can only infer that the time of opacification corresponds to the age level of the cataract in the lens.

The reduplication cataract illustrates in another way how an opacity of the lens may serve as a marker of time. Here the opacity of the capsule is separated from its imprint by a zone of clear fibers, and the depth of this clear lamella is proportionate to the length of time that has elapsed between the formation of the cataract and the date of examination.

As a final word, it must be remembered that a congenital defect does not necessarily refer to one bound up with the embryologic changes of the lens, but simply to a defect present at birth. An example of this is the congenital anterior polar cataract which is caused by a perforation in utero of the cornea instead of by an error in growth. It must also be borne in mind that the term "developmental," strictly speaking, covers the entire span of life, for the lens never ceases to develop new fibers. One cannot, therefore, confine the application of this term to the prenatal period. Lastly, it must be remembered that any opacity outside of the early layers of the fetal nucleus cannot have been existent at birth, and is therefore not congenital.

Professor von Lenhossek and his assistant Dr. Mihalik placed at my disposal their collections of embryos and gave me permission to photograph certain of their sections.

BUPHTHALMOS SECONDARY TO CONGENITAL IRIDOCYCLITIS *

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MONTREAL, CANADA

The pathologic changes that I shall describe were found in a buphthalmic eyeball, which was sent to me by the late Dr. Sprague, of Barre, Vt., with the following notes:

William B., aged 14 years. Right eye always blind. Whiteness of the pupil, as well as some enlargement of the globe, noticed by the nurse and by the mother at birth. Eye painful at night for the past three months. Globe markedly buphthalmic and tender to the touch. Lids closed with difficulty. Conjunctival and episcleral congestion. Cornea clear; iris bombé. Pupil, normal in size and fixed (no response to mydriatics), occupied by dense organized exudate. No fundus reflection. V. = no p. l.; T. = N. Left eye, normal; V. = 6/6 with a + 0.25 sphere.

The patient was the sixth of a family of nine children, none of whom was similarly affected. Syphilis could be excluded.

The globe was received at the laboratory of the Royal Victoria Hospital in a collapsed condition, having been forwarded in water on a hot day. However, Miss Holland, a technician, succeeded in obtaining satisfactory celloidin sections, after fixation in formaldehyde. The following measurements necessarily understate the dimensions of the eyeball during life: vertical, 27.5 mm.; horizontal, 30 mm.; anteroposterior, 32 mm.

Microscopic Examination.—The conjunctiva had been cut close to the limbus, but what remained seemed to extend onto the cornea farther than normally. The cornea appeared normal throughout, except at the periphery, where the anterior half of the substantia propria showed vascular and perivascular infiltration and imbibition.

The sclera (fig. 1) showed general thinning, particularly in regard to the ciliary and intercalary regions. Because of the atrophy of the scleral fibers in this region, Schlemm's canal was nowhere visible. There was pronounced cupping of the optic disk, in association with complete atrophy of the optic nerve and changes suggestive of a myopic crescent-rounding of the sclera on the temporal side of the cup, absence of the overlying choroid and retinal pigment epithelium and widening of the corresponding subarachnoid space. The veins over the whole surface of the sclera and about the optic nerve were markedly dilated.

In the pupillary area and for a few millimeters on either side, the anterior capsule of the lens (fig. 2 *A*), was covered by and intimately adherent to a thick layer of fibrous connective tissue, which stained deeply by the van Gieson method. The iris was everywhere adherent to this membrane, forming a complete annular synechia. It was apparently the anterior surface of the tip of the iris (fig. 2 *B* and B_1) rather than the posterior surface that adhered to the exudate, and the stroma of the iris (fig. 2 *C*), which was denser and more deeply pigmented,

* Submitted for publication, April 20, 1931.

extended with diminishing thickness onto the surface of the underlying occluding membrane.

The pupillary exudate was laminated, some of the fibers being cut in cross-section (fig. 2 *D*), while others (fig. 2 D_1) were sectioned longitudinally. Very few nuclei were observable. A single layer of endothelial cells, continuous with those of the iris, covered the anterior surface of the membrane.

The pigment layer (fig. 2 *E*), which was greatly thickened, was reflected from the posterior surface of the iris onto the anterior surface of the lens (exudate),

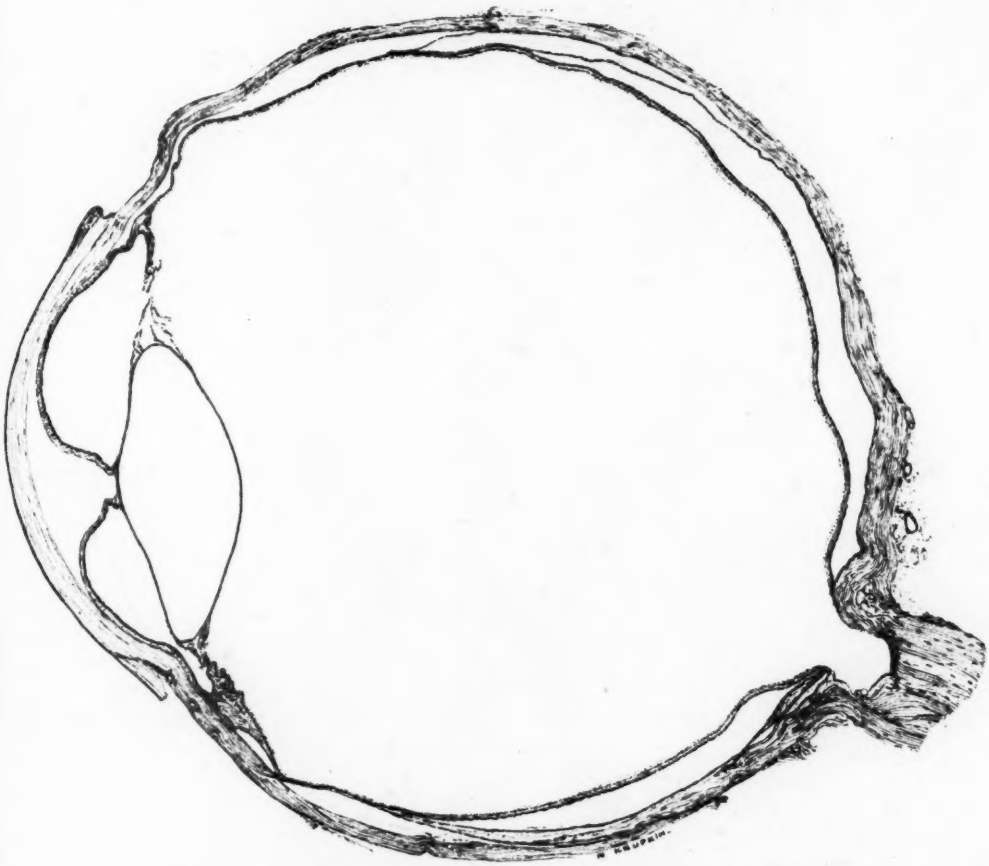

Fig. 1.—Drawing showing the thinning of the sclera and the changes in the anterior segment of the globe and the optic nerve.

along which in some sections it passed outward for a considerable distance. In numerous sections, in the angle formed by the junction of the posterior surface of the iris with the anterior surface of the lens (exudate), one could see cryptic spaces (fig. 2 *F*) of irregular size and shape, formed by the separation of the two rows of cells of the pigment layer of the iris.

From the lens, the iris, which was greatly thinned, lengthened and atrophic, passed forward and outward, in typical bombé fashion, to fuse intimately with the inner surface of the cornea and the sclera, beyond the termination of Descemet's membrane (fig. 1).

The ciliary body was everywhere reduced to a coarse meshwork of fibrous tissue. Its muscle fibers had largely, if not entirely, disappeared, and its anterior surface, which was held inward by the zonular fibers, was deeply cupped as a result of the increased intra-ocular pressure (fig. 1). The choroid was greatly thinned; in some sections it was reduced to a mere thread, and it showed no gross hyaline changes. The latter was also true of the eye as a whole.

Owing to numerous artefacts, observations on the retina and the lens were valueless. The kinking of the capsule of the lens at both sides of the exudate (fig. 2 *G*) might be attributed to antemortem contraction of the membrane, or might represent an artefact.

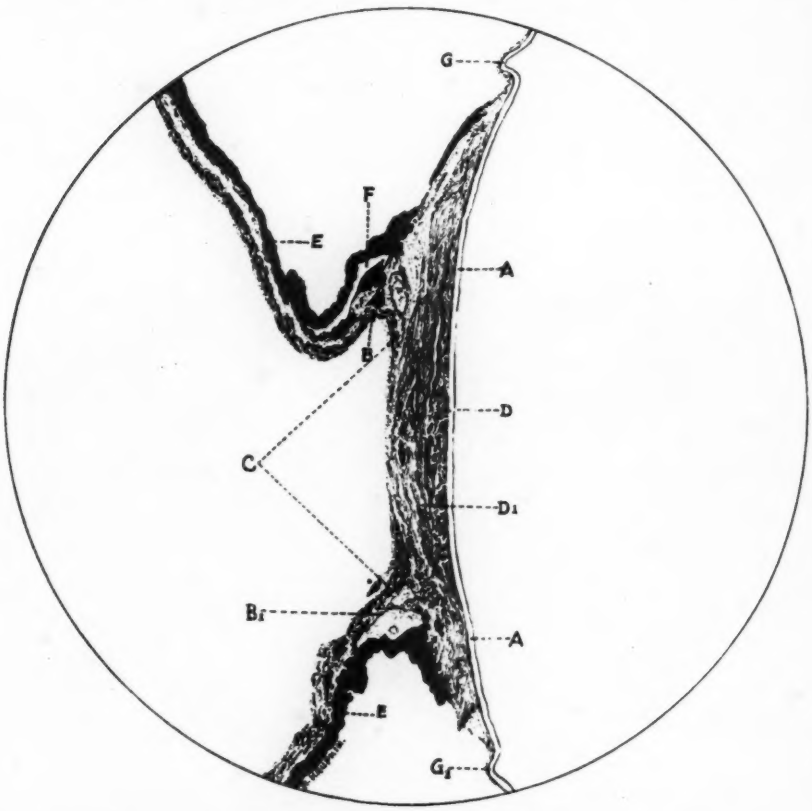

Fig. 2.—The pathologic changes in and near the pupil. *A* indicates the anterior capsule of the lens; *G* and G_1, kinks in the anterior capsule; *D*, connective tissue fibers in cross-section; D_1, the connective tissue fibers in longitudinal sections; *E*, the iris, the tip of which is adherent to the connective tissue deposit at *B* and B_1; *C*, connective tissue extending from the iris onto the anterior surface of the deposit; *F*, cyst formed by the separation of the cells of the pigment layer.

COMMENT

The pathologic changes I have noted are typically those seen in chronic (untreated) iritis. As a reasonable explanation of this case cannot be found in a developmental aberration and the pupillary deposit

and bulbar enlargement were present at birth, a diagnosis of congenital buphthalmos secondary to prenatal iritis must be made. I can find no similar case reported in the literature, though the occurrence of prenatal iritis is widely recognized.

Dr. N. Krupkin supplied the two excellent drawings.

CONJUNCTIVITIS AND BLEPHARITIS DUE TO YELLOW MERCURIC OXIDE *

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As described by Fuchs [1] and Fox,[2] an individual susceptibility or idiosyncrasy occurs in drug conjunctivitis. This may follow preparations like mild mercurous chloride and yellow mercuric oxide, which are used in the treatment for conjunctivitis eczematosa and may produce an irritative conjunctivitis and involvement of the surrounding skin. Fox [2] found that particularly the prolonged use of cocaine and atropine and occasionally physostigmine and homatropine resulted in a follicular catarrah of the conjunctiva and considerable swelling of the lids. Duane [1] compared this susceptibility to drugs with hay-fever sensitivity. He believed that in susceptible persons, following the initial contact of the conjunctiva with pollen granules an inflammation occurs. This susceptibility is increased by the patient's having had one attack of the disease, and later contact of the conjunctiva, even with minute quantities of the pollen, causes a recurrence of the disease. Similarly, drugs, not poisonous to the majority, when instilled into the conjunctiva or applied to the lids, cause an inflammatory reaction in unusually susceptible people. Their tissues, having once been affected by the drugs, are sensitized or put into a state in which they react more readily to the poison than before.

Galewsky [3] used a 1 per cent solution of procaine hydrochloride as a collyrium during attacks of seasonal hay-fever, without ill effects. The following year, after using this solution for four days he developed a marked inflammation of the eyes and face. When he recovered, he confirmed this belief in an acquired sensitivity to procaine hydrochloride by reapplying the solution first to the conjunctiva and the second time to the nasal mucous membrane. Within a few hours

* Submitted for publication, May 22, 1931.

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1. Fuchs, E.: Text Book of Ophthalmology, ed. 5, trans. by Duane, Philadelphia, J. B. Lippincott Company, 1917.

2. Fox, L. W.: Practical Treatise on Ophthalmology, New York, D. Appleton & Company, 1910.

3. Galewsky: Ueber Novokaindermatitiden bei der Anwendung von Novokain Augenwasser, Dermat. Wchnschr. **85**:1195, 1927.

marked local inflammatory reactions developed. Kesten and Laszlo [4] reported a case of blepharitis and dermatitis of the face occurring in a surgeon when a 1 per cent solution of procaine hydrochloride was accidentally squirted on these areas. This patient's skin was found sensitive to the local application (patch test) of a 1 per cent solution of procaine hydrochloride. Occurrences of conjunctivitis and dermatitis, following the use of atropine to induce mydriasis, are reviewed by Biberstein.[5] This work also includes three patients who developed exanthems following the instillation of atropine into the conjunctival sac. On local passive transfer of this sensitivity, by their serum, an erythematous reaction developed. The skin of one of the patients was shown to be sensitive to atropine sulphate by patch and intradermal tests. Among the drugs used in ophthalmotherapy, Biberstein [6] found patients sensitive to mercury, atropine, a proprietary bismuth preparation, cocaine, procaine hydrochloride and scarlet red. The sensitivity was manifested by an inflammatory reaction of the conjunctiva or skin, or both. In three of these patients, who developed an inflammatory reaction following the application of mercury oxycyanide, atropine sulphate and cocaine, respectively, the skin sensitivity was confirmed by patch tests. Cummer [7] reported a case of dermatitis of the eyelids due to the prolonged use of ethylmorphine hydrochloride, U. S. P. Although a patch test with the drug, which was evidently applied to a nonsensitized skin area, gave negative results, the dermatitis disappeared following its use and recurred on its resumption.

Because of the infrequency with which conjunctivitis, blepharitis and dermatitis follow the repeated use of as commonly prescribed a medicament as yellow mercuric oxide and the ease with which the cause of the inflammatory reaction may be detected, the following case is reported.

REPORT OF A CASE

J. E., an 8 year old school girl, entered the clinic on April 23, 1931, complaining of an inability to see well and swelling of the face of thirty-six hours' duration. A week previously, following an acute coryza, the child complained of "watering of the eyes." As this complaint persisted, zinc sulphate eye drops and 0.5 per cent yellow mercuric oxide were prescribed. Twelve hours after the instillation of the drops and application of the salve, the mother noted a beginning redness and swelling of the eyelids. As the child did not complain, these medicaments

4. Kesten, B., and Laszlo, E.: Dermatitis Due to Sensitization to Contact Substances, Arch. Dermat. & Syph. **23**:221 (Feb.) 1931.

5. Biberstein, H.: Zur Atropinüberempfindlichkeit, Arch. f. Dermat. u. Syph. **154**:555, 1928.

6. Biberstein, H.: Ueberempfindlichkeiten in der Ophthalmotherapie, Ztschr. f. Augenh. **71**:283, 1930.

7. Cummer, C. L.: Dermatitis of the Eyelids Caused by Dionin, Arch. Dermat. & Syph. **23**:68 (Jan.) 1931.

were reapplied twice during the next twenty-four hours. The child was brought to the clinic the following morning because the swelling had spread and her eyes had begun to itch and burn. Except for the face the general condition was normal. The affected areas included the low half of the forehead, the eyes, lids and upper third of the cheeks and nose. There was a watery secretion from both eyes and a marked swelling of the palpebral conjunctiva with moderate injection of the vessels and intense edema of the eyelids; the involved skin, which was crimson, smooth, shiny and diffusely edematous, with a local increase in temperature, was sharply demarcated from the surrounding normal skin. Except for the lack of constitutional symptoms the lesions were strikingly like erysipelas. They were treated with iced wet dressings of boric acid, and the eye drops and ointment were discontinued. In three days the conjunctivitis and blepharitis had almost disappeared and the inflammation of the skin had regressed somewhat,

A B

Fig. 1.—*A*, second attack of conjunctivitis, blepharitis and dermatitis following the application of yellow mercuric oxide to the eyelids. *B*, first attack of blepharitis and dermatitis (three years previously) following eye drops and the application of a yellow ointment to the eyelids.

leaving a minute vesicular eruption, as shown in figure 1 *A*. Calamine lotion was then applied to the involved area, and in two weeks the condition had completely healed.

The past history of the patient was of interest in that three years previously she had an "inflammation of the eyes," and following the application of drops and "the same kind of yellow ointment" similar but more intense lesions had developed. This inflammation appeared only after use of the medications for several weeks. When the eruption had persisted a month or so, the child was referred to the skin clinic. The photograph (fig. 1*B*) taken at that time (January, 1928) shows the previous blepharitis and dermatitis. The eye medications were discontinued,

and the lesions cleared rapidly. From that time to the present illness the patient applied no medications, and the skin remained normal. The patient gave no history of sensitivity to other drugs or of urticaria, eczema, hay-fever, asthma or migraine. She had not had any disease (such as diaper rash, impetigo, warts or syphilis) for which mercury preparations might have been prescribed. She had received no drugs containing iodine.

The problem then presented itself of determining to which drug the patient was sensitive. This was done by means of the patch test.

Proof of Sensitivity to Mercury.—In inflammatory reactions of the skin due to external sensitization (dermatitis venenata), the cause of the eruption may be determined frequently by the application of patch tests (Jadassohn[8]). This consists in applying the substance thought to be the cause of the eruption to the intact skin in a concentration not irritating to normal skin. In making the patch, the center of a 4 cm. square of adhesive tape is protected with a 3 cm.

Fig. 2.—Positive patch test to 0.5 per cent yellow mercuric oxide (at left) and negative patch test to zinc sulphate eye drops (at right).

square of "cellophane" or "silkoid" (to prevent an adhesive tape dermatitis). On the center of this is placed a 1 cm. square of four-ply gauze which is well covered with the substance to be tested. These "patches" are stuck on the normal skin for twenty-four hours. A patch test is considered positive when a well defined, inflammatory area covered with papulovesicles, vesicles or a bulla occurs at the test site. Almost all of the positive reactions are accompanied by pruritus. In most instances, the positive reaction is present at the end of twenty-four hours. A positive patch test indicates that the epidermis is sensitive to the substance applied and that a sensitization reaction (dermatitis) may develop or has developed following contact with this substance.

8. Jadassohn, J.: Hautkrankheiten bei Stoffwechselanomalien, Internat. Dermat. Kong. Verhandl. u. Ber. **2**:231, 1904; Bermerkungen zur Sensibilisierung und Desensibilisierung bei den Ekzemen, Klin. Wchnschr. **21**:1680 and 1734, 1923.

In the patient under observation, patch tests were first applied with the zinc sulphate drops and the 0.5 per cent yellow mercuric oxide ointment which the patient had been using. In twenty-four hours there was a definite positive reaction to 0.5 per cent yellow mercuric oxide. The test with zinc sulphate was negative. These are shown in figure 3. To determine if a sensitivity existed to other mercurial preparations, patch tests were then applied using the following: corrosive mercurous chloride, 0.05 per cent; mercury salicylate, 0.06 per cent; metallic mercury, 1 per cent; ammoniated mercury, 5 per cent, and a second test with 0.5 per cent yellow mercuric oxide. All reactions were positive, the one to 0.5 per cent yellow mercuric oxide being the most marked. The positive patch tests with the last two mercury compounds are shown in figure 4. Patch tests with the foregoing substances gave negative results on normal persons. By the positive patch

Fig. 3.—Positive patch tests to 5 per cent ammoniated mercury (above) and to 0.5 per cent yellow mercuric oxide (below).

tests the sensitivity of the skin to mercury was demonstrated. No focal reaction followed these positive tests.

In dermatitis due to external irritants it is usually impossible to demonstrate antibodies in the blood, although Biberstein [5] believed that he detected them in a patient sensitive to atropine. In the case reported, their demonstration was attempted by a modified Prausnitz-Küstner [9] method. This consists in passively transferring the antibodies in the blood serum of a sensitized person to a normal person. The antigen (or sensitizing substance) is then applied to the site where the sensitive person's serum was injected. If a systemic sensitivity exists in the sensitized person, a reaction like that found in the positive patch test occurs at

9. Prausnitz, C., and Küstner, H.: Studien über die Überempfindlichkeit, Zentralbl. f. Bakteriol. **86**:160, 1921.

the test site in the normal subject. The blood serum of the patient reported was tested, by the aforementioned method, on four patients (two with and two without allergic symptoms) with negative results.

COMMENT

It has long been known that conjunctivitis and dermatitis may follow the application of certain medications, but this has frequently been thought of as an irritating or poisonous quality rather than sensitization. The literature and the case reported show that the normal epidermis may be sensitized by the repeated application of a number of nonprotein substances which are not primarily irritating or toxic to the skin. As pointed out by Biberstein [5] this inflammation of the mucous membranes and skin may occur either after the first or after prolonged application of a medication. In the case reported, there was no history of the internal or external use of mercury until several weeks before the onset of the first eruption, i. e., three years before the present illness. At this time there was a history of the application of a yellow ointment over a definite period with a resultant inflammatory reaction and a disappearance of the inflammation after the medication was discontinued. From this story one assumes that the patient, by repeated application of yellow mercuric oxide to the eyelids, sensitized the epidermis to mercury and that the resultant inflammatory process was the manifestation of an external skin sensitization to an external contact. This method of the mechanism of sensitization is further emphasized by the recurrence of symptoms three years later following a similar medication which contained mercury, the production of a localized dermatitis on the application of mercurial preparations to the normal skin (the positive patch tests) and the inability to demonstrate circulating antibodies in the blood serum. The opposite of this method of sensitization (i. e., an internal reaction to internal sensitization) may occur in eczema and urticaria due to foods, and here one does not usually obtain a reaction when the sensitizing substance is applied to the unabraded skin (i. e., the patch test is negative) but does find circulating antibodies in the patient's blood serum. The positive patch tests and the negative passive transfer test indicate a sensitization of the skin only to mercury. Although the ingestion or injection of mercurial preparations might cause a recurrence of the dermatitis and conjunctivitis (i. e., an internal reaction to external sensitization) it is quite possible that the patient might be desensitized to mercury by either of these methods, as with poison ivy dermatitis.

The patch test is a convenient method of testing for the presence of epidermal sensitivity. It is a less heroic measure than the resumption of the suspected drug for a proof of sensitivity. If the sensitivity of

the epidermis is generalized, as in the case reported, the patch test should be positive regardless of where it is applied. If, on the other hand, the sensitivity is localized, as in Jadassohn's [8] and Cummer's [7] cases and some of those reported by Kesten and Laszlo,[4] it will be necessary to apply the patch test to the area that has been sensitized.

SUMMARY

A case of conjunctival and skin sensitivity to yellow mercuric oxide is reported.

This sensitivity was confirmed by a disappearance of the inflammation when mercury ointment was discontinued, by a reappearance of the eruption following its application and by positive patch tests with mercury.

Ophthalmologic Review

TUMORS IN THE PITUITARY AREA

THE NEUROLOGIC SYMPTOMS *

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PHILADELPHIA

Knowledge of the symptomatology of tumors and of other lesions in the pituitary region has increased enormously in the last two decades, and the methods of diagnosis are such that, because of the information gained first from the ophthalmologic and, secondly, from the roentgenologic standpoint, the general diagnosis of tumors in this area is comparatively easy. On the other hand, knowledge of the precise causes of the so-called endocrine disturbances, which at times are the sole manifestations of lesions in this area, has not advanced as rapidly, for while ten or more years ago it was customary to ascribe such symptoms to overactivity or underactivity of the anterior, middle and posterior lobes of the hypophysis, a great deal of doubt has been cast on such theories, and recently it has been the fashion to ascribe such symptoms to disturbances in the parts above the hypophysis, that is, in the walls of the third ventricle, in the hypothalamic region, the infundibulum and the stalk that connects the infundibulum with the posterior hypophysis. In fact, this tendency has been manifested to such an amazing degree that, while it is generally acknowledged that gigantism and acromegalia result from overactivity of the anterior pituitary lobe, there are some excellent investigators who ascribe the locale of this symptom complex to the hypothalamus.

Experience with pituitary lesions points to one outstanding fact, and that is that in the diagnosis of tumors in this area not only the pituitary gland itself must be considered, but all of the aforementioned surrounding structures. In this region should be included the sphenoid sinus, lying underneath the sella, from which adenomas occasionally are derived.

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* Submitted for publication, April 24, 1931.

* Read by invitation before the College of Physicians, Philadelphia, Section on Ophthalmology, Dec. 18, 1930.

* One of a series of papers in a symposium on the "Symptomatology of Pituitary Tumors." In this paper only the neurologic symptoms are discussed. The ophthalmologic, roentgen and surgical aspects are discussed separately by other authors. No literature is quoted, but it was used extensively.

FUNCTIONS OF THE PITUITARY AREA

At this juncture it would be well, if it were possible, to summarize the function of the various components of what could be called the "pituitary area." Experimental evidence seems to indicate rather definitely that so far as the anterior lobe is concerned there are two definite hormones, one concerned with the growth principle, and the second, with the sex principle. While the hyperpituitary symptoms are distinctive from the hypopituitary, nevertheless, they both have this in common: they produce demonstrable regressive changes in the reproductive organs, for in both hypopituitary and hyperpituitary states there is an inhibition of the ovarian cycle.

So far as the function of the posterior hypophysis is concerned, it would seem advisable not to consider it separately, but as part of a mechanism including the posterior lobe, the stalk, infundibulum and the adjacent hypothalamic structures, for it is impossible to differentiate clinically lesions in any one of these separate structures.

There is some anatomic evidence of interrelation of these parts, for nonmedullated nerve fibers have been traced from the supra-optic and paraventricular nuclei of the subthalamic region, converging at the infundibulum, and passing in a compact bundle to the posterior stalk to enter the posterior lobe and the pars intermedia. Furthermore, the enveloping structures of the posterior lobe, as well as the stalk and infundibulum, have similar epithelial characteristics, and there is some evidence that these cells represent the secretory principle which acts on all these inclusive structures. Therefore, it can be assumed that the pars intermedia, the posterior lobe, the stalk and the infundibulum have a somewhat similar physiology.

The diencephalon, or interbrain, has been receiving increasing attention. It is one of the oldest portions of the brain and remains essentially unaltered in all creatures that have a brain. It has already been mentioned that there is a connection between certain nuclei in the walls of the third ventricle with the posterior and middle portions of the hypophysis. The infundibular lobe is a downward extension of the diencephalon. Herein are supposed to lie ancestral mechanisms common to all species, which have to do not only with the vegetative functions but with their primitive instincts as well, such as mating, satisfying hunger and thirst, restoration from fatigue by sleep, regulation of body temperature, self-protection by combat, etc.

Furthermore, there is experimental and clinical evidence that the cerebral sympathetic centers are in the hypothalamus. Nonmedullated fibers have been traced to the spinal cord which connect with the abdominal and thoracic viscera, and there is some evidence that some of these fibers connect with the anterior hypophysis itself.

CLINICAL EVIDENCE

In the last few years a vast amount of literature has accumulated on the symptomatology of lesions within the third ventricle. In 1911, I reported twenty-seven cases, this being the largest study of lesions in this area up to that time. As this area is compressed and sometimes directly implicated by pituitary tumors, the function of this part is important in this connection.

The walls of the third ventricle consist of the optic thalami on either side. The thalamus is a sensory organ; all sensory impulses from the periphery converge here and are reintegrated, and from there go to the cortex. Lesions in this area may give so-called central pains. The presence of sympathetic centers has already been mentioned. In addition, from encephalitic lesions, which frequently implicate this part of the brain, considerable knowledge has been gained. Suffice it to say that as a consequence of lesions in the third ventricle there is disturbance in the sleeping rhythm, usually hypersomnia; alterations in water metabolism, causing diabetes insipidus; disturbances in fat metabolism, causing obesity; disturbances in growth, causing infantile sexual states; lastly, vegetative and emotional activities of the utmost importance.

More specifically it has been proved that diabetes insipidus is produced by minor lesions of the hypothalamic region, and that it can be assumed that when this is present the infundibulum is implicated rather than the hypophysis. It can also be assumed that obesity is consequent to lesions in the hypothalamus.

In the last few years a great deal has been written on so-called pituitary cachexia, but here also the evidence seems to be that it is consequent to lesions of the hypothalamic region, and not of the pituitary gland.

Disturbance of sleep as a symptom is of the utmost importance, provided it occurs before the development of other symptoms of tumor of the brain or, if late, in cases in which there is no increase of intracranial pressure. Such stupor should be distinguished from the general sluggishness and torpor that are sometimes present in any space-taking lesion of the brain.

One of the outstanding groups of symptoms in lesions of the third ventricle is the disturbances in emotional states and in mentality. Frequently there are violent emotional crises; often acute psychoses; more commonly, however, there are torpor and lethargy.

Much has been written of disturbances of carbohydrate metabolism, but evidence points to the fact that while glycosuria occurs rarely in acromegalia, it does not accompany tumors of any other sort. Its presence in tumors of the brain is not definite.

To summarize, if accompanying hypersomnia there occur diabetes insipidus, adiposity and genital dystrophy, a lesion of the hypothalamic region should be suspected. These symptoms may occur simultaneously or successively, or in any combination.

On the other hand, primary hypophyseal tumors, not projecting into the third ventricle, may cause apathy and lethargy from lowered metabolism.

From this hasty sketch it is obvious that there is little precise knowledge of the functions of the pituitary area. A review of the literature leaves one confused. One reason for this is the lack of definite knowledge of the functions of the so-called ductless glands and confusion as to what is produced by irritative and destructive lesions. In the discussion, for example, of the physiology of water metabolism in relation to diabetes insipidus, it is sometimes assumed that different symptoms follow irritative and destructive lesions of the same structure. From the neurologic standpoint, this is impossible, for a dead organism cannot produce symptoms. It would be a more proper assumption that whatever symptoms are present in the development of any lesion are the result of the activity of the unimpaired structures. So far as the endocrine glands are concerned, there is always an assumption that one gland may compensate for the inactivity of the other. This also is impossible. It is obvious that the hormone of the ovarian gland, for example, cannot be secreted by any other organ. It is clear, however, that there is a governing relationship, for there is some evidence that activity of the basophilic cells in the anterior lobe of the pituitary gland bears a definite relationship to the ovarian cycle, and in any lesion of the anterior pituitary which destroys these cells, amenorrhea becomes apparent at once.

No attempt is made in this paper to describe the symptoms of lesions in detail. On the other hand, there are certain general facts that can be mentioned. The most common symptom in women is disturbance of the menstrual cycle and of libido in men. This is present in 80 per cent of cases and may be of extremely long duration. One case is reported in which amenorrhea had been present for twenty-nine years. Headache is an early symptom and is usually frontal, but may be referred to one eye. Slight fatiguability is common. Endocrine symptoms, of which there are legion, are present early. No general discussion of the endocrine symptoms will be attempted; I shall only point out that they are largely dependent on the age of the patient at the onset of the lesion. If the lesion occurs during childhood or adolescence, the syndrome of infantilism is apparent. This is characterized by hypoplasia of the genital organs and the absence of secondary sexual characteristics in a person past the age of puberty. With the general inhibition of growth there are segmental deposits of fat in the abdomen, pelvis, shoulders, neck and breasts, the distal extremities appearing normal.

If the lesion begins in the adult, there are usually disturbance of the menstrual cycle in women and impotence in the male with regression in the genital organs, and sometimes in the whole sex morphology. Obesity is usually present.

PITUITARY TUMORS

The tumors of the pituitary region may be divided into (1) intrasellar and (2) extrasellar. The intrasellar tumors are composed in the great majority of instances of the adenomas which have their origin in the glandular cells within the anterior lobe of the pituitary body. These glandular cells are of three types, depending on their staining reaction in response to certain specific differential stains; the eosinophil, basophil and chief cells. The eosinophilic cells are by far the most numerous and contain medium coarse granules which stain an intense pink. The basophilic cells are less numerous and contain coarse granules which take the basic stain on differential dyeing. These eosinophilic and basophilic cells have been called alpha and beta cells, respectively. The chief cells are common cells of the pituitary and take no specific stain; that is, they remain unstained by the usual differential methods of staining the specific granules in the pituitary body.

The adenomas which are derived from the pituitary have their origin in one of these three types of cells. There is, first, the chromophile adenoma, which is composed chiefly of eosinophilic cells and is associated in practically every instance with the clinical picture of acromegalia. It is, in other words, the type of adenoma found in the hyperpituitary syndromes due to tumor. There is in addition the basophilic adenoma, which is a rare type, only a few instances having been recorded in the literature. It can be dismissed because as yet extremely little is known concerning the relationship of this type of tumor to specific clinical syndromes. It is probably concerned with reproductive processes.

The third and probably the most common type of adenoma in the sella is the chromophobe, so called because the cells composing this tumor do not take up a specific dye similar to the eosinophilic staining properties of the chromophilic adenoma, for example. This type of tumor is derived from the chief cells of the pituitary, these cells in their normal state having no specific granules that can be stained by ordinary methods. The chromophobe adenoma is associated clinically with the hypopituitary syndromes. It is found in association with the tumors that cause endocrine disturbances, such as dysmenorrhea or amenorrhea, pathologic deposits of fat, diabetes insipidus and other well known syndromes associated with hypopituitarism, so that it is possible to say that the types of primary pituitary symptoms are found in association with definite types of pathologic pituitary processes, that is, so far as the symptoms are caused by primary pituitary tumors.

The second type of intrasellar tumor is that of Rathke's pouch. This tumor is either intrasellar or extrasellar. It is difficult to estimate the exact percentage of cases in which it is intrasellar, but the remnant of Rathke's pouch is an intrasellar structure, and it is therefore true that in a number of instances Rathke's pouch tumors, before they grow to the great size which they subsequently attain, are intrasellar neoplasms. They are derived from the remnant of the ingrowth from the pharyngeal epithelium and from the anterior portion of the pituitary body. They differ markedly in their histologic structure from primary pituitary adenomas. They are composed of epithelial cells often arranged in stratified form and often with rosettes into which they project cilia from the cells of the buccal mucosa. These tumors occur in childhood, and they are associated with definite endocrine disturbances. In the

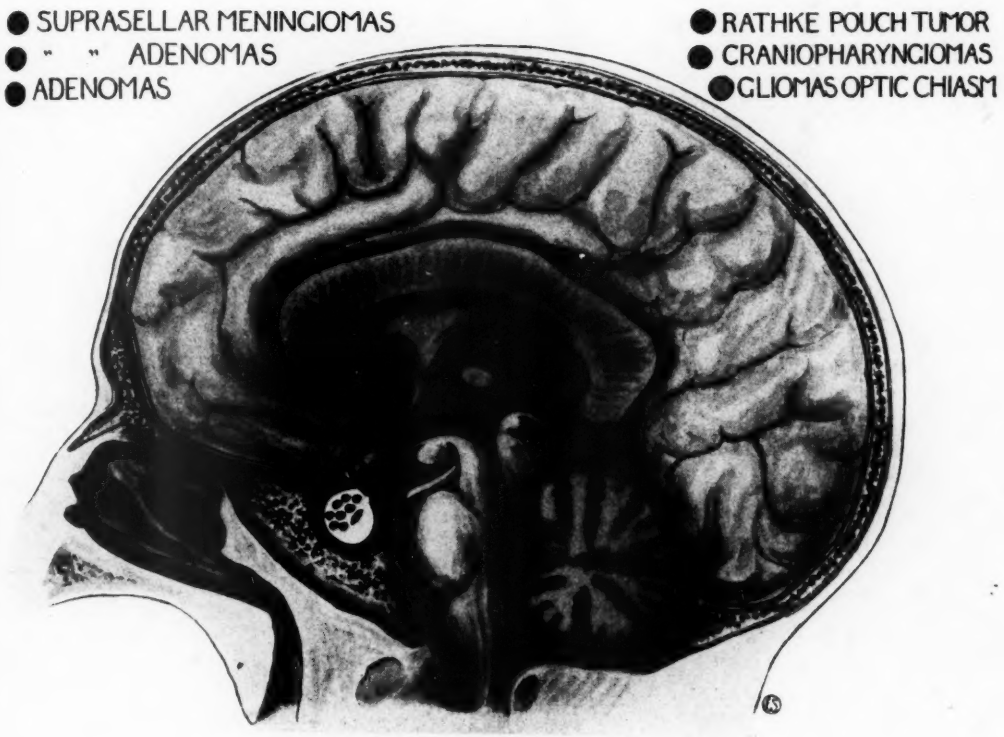
Intrasellar Pituitary Tumors

General Scheme			
Intrasellar	Adenomas	Chromophile Chromophobe Mixed	
	Rathke's pouch		
General Class	**Type**	**Pathology**	**Clinical**
Adenomas	Chromophile	Derived from and composed of eosinophilic cells of anterior pituitary	Always associated with hyperpituitary syndrome-acromegalia or gigantism
	Chromophobe	Most common type of pituitary tumor composed of the chromophobe elements of anterior pituitary, viz.: the chief cells	Associated with the hypopituitary syndrome—depression of sexual function, adiposity, decrease in metabolic rate, infantilism, etc.
	Mixed	Contains both chromophobe and chromophile elements	Associated with clinical evidences of hyperpituitarism and hypopituitarism
Rathke's pouch tumors		Originate in sella and expand beyond it, or usually begin as extrasellar growth derived from anlage of pharyngeal pouch; are epithelial tumors	Occur in young, usually at puberty, tumor calcified in 80 to 85 per cent; hypopituitary syndrome and diabetes insipidus common

great majority of cases, from about 80 to 85 per cent, they are calcified. Their size varies considerably from that of a small nut to the size of a baby's fist. While it is true that in some instances the tumor is intrasellar, it grows and usually becomes extrasellar, so that when the patient is examined the signs are those of an extrasellar tumor. Little is known about the roentgenologic differentiation of an intrasellar adenoma from an intrasellar tumor of Rathke's pouch. Presumably they produce similar kinds of enlargement, but this is not certain.

The suprasellar tumors are of various types and give rise to what has been termed the chiasmal syndrome. These tumors may be listed as follows:

(1) Suprasellar adenomas.
(2) Suprasellar meningomas.
(3) Tumors of the craniopharyngeal duct.
(4) Gliomas of the optic chiasm.
(5) Suprasellar aneurysms.



Tumors of the pituitary region.

In their early stages the suprasellar tumors give rise to what has been referred to as the chiasmal syndrome, namely, primary optic atrophy, bitemporal hemianopia and a normal or relatively normal sella turcica in contradistinction to the markedly enlarged and ballooned sella turcica found in the primary pituitary tumors. The suprasellar adenomas are derived in one of two ways; either they occur from very superficially placed pituitary tissue, which extrudes itself early in the development of the tumor through the diaphragm of the sella and becomes suprasellar, or else they are derived from embryonic rests along the infundibular stalk. In some instances they are derived from the normal accessory pituitary gland which lies in the sphenoid bone or in the roof

Suprasellar Pituitary Tumors

General Scheme		
Suprasellar	Meningiomas Adenomas Craniopharyngiomas Gliomas of optic chiasm Aneurysms	Chiasmal syndrome Primary optic atrophy Bitemporal heminanopia Normal or relatively normal sella turcica

Class	Pathology	Clinical
Meningioma	Typical meningioma structure; arise from tuberculum sellae	Occur in middle-aged, normal sella; produce endocrine disorders rather late due to expansion
Adenoma	Derived from anlage along infundibulum or within sella, escaping through diaphragm	More pressure atrophy of sella; mild hypopituitary signs; more rapid in course than meningiomas
Craniopharyngioma	Includes craniopharyngeal duct tumors and adamantinomas; always suprasellar; derived from epithelial rests along infundibular stalk	Often calcified; frequently cause secondary pituitary and hypothalamic symptoms; endocrine disturbances very common
Gliomas of Chiasm	Arise from chiasm or adjacent wall of III ventricle and spread along nerve, even into nerve head	Occur in young; field cuts irregular; sella has pear-shaped forward expansion
Aneurysm	Give history of severe sudden headache, unconsciousness and blood in spinal fluid	

of the pharynx and which in the formation of the tumor grows up through the sphenoid sinus and becomes an extracranial neoplasm giving the picture of a suprasellar tumor. These suprasellar adenomas give rise to endocrine disturbances, the nature of which varies considerably in accordance with the area involved. They produce no enlargement of the sella, but they tend to erode the clinoid processes.

Suprasellar meningiomas also give rise to the chiasmal syndrome. They originate from tuberculum sellae. They occur in middle-aged persons and produce practically no deformity of the sella.

The tumors of the craniopharyngeal duct are derived from epithelial rests which in the normal person are found lying along the infundibular stalk in the angle of its attachment to the tuber cinereum and in the angle of its attachment to the hypophysis. These tumors are therefore

purely suprasellar tumors, and are probably more common than they have been thought to be. Their histologic structure is characteristic; they are epithelial tumors arranged in cords or papillae with an appearance similar to adamantinomas.

The gliomas of the optic chiasm occur in younger persons and produce a chiasmal syndrome. The hemianopia that they produce is of a much more irregular type than that produced by other tumors. In other words, the infiltrating glioma spears a good many fibers in its growth and does not give rise to the cleancut hemianopia seen in the meningiomas, for example. They often involve the optic nerve directly and press forward through the optic foramen, producing an enlargement of the opening which can be detected by the x-rays and which produces a sella deformation by its dilatation, without, however, enlarging the sella.

DIFFERENTIAL DIAGNOSIS

In addition to the intrasellar and suprasellar tumors that have already been discussed, consideration must be given to two facts: 1. Tumors of the pituitary may give rise to symptoms of lesions elsewhere, by direct extension into distant parts, such as the cerebellar region, by extension from the third ventricle to the lateral ventricles, or, what is most common, by extension into the aqueduct of Sylvius, causing symptomatology referable to the midbrain, which is chiefly confused with symptoms produced by cerebellar lesions. 2. Instead of direct extension of the tumor, a generalized internal hydrocephalus may be produced, with dilatation of the lateral, third and fourth ventricles, and attending phenomena.

On the other hand, tumors of the cerebellum frequently cause symptoms of dyspituitarism, because of the secondary internal hydrocephalus, dilatation of the lateral and third ventricles and direct pressure on the pituitary region. The differential diagnosis, however, should not be difficult if an adequate history of the progress of the symptoms is obtained, for in lesions encroaching on the pituitary area, the symptoms referable to this part appear late. Again, there are well recorded instances of generalized internal hydrocephalus with consequent ballooning of the third ventricle and pressure on the pituitary region. In such cases diagnosis cannot be made until the postmortem examination, but here again the history, if it is available, should give a clue to the diagnosis.

News and Notes

EDITED BY DR. J. HERBERT WAITE

GENERAL

Tuberculosis Convention.—A tuberculosis convention will be held at Davos, Switzerland, Oct. 5 to 10, 1931. The subject has been divided into three parts: Development and Course of Pulmonary Tuberculosis, Extrapulmonary Tuberculosis and Collapse Therapy. Among the papers in the section dealing with extrapulmonary lesions, the following will be of interest to ophthalmologists: Ocular Tuberculosis in the Picture of General Tuberculous Disease, Dr. E. Werdenberg, Davos; Experiences in Ocular Tuberculosis in Their Relation to General Tuberculosis, Professor Schieck, Würzburg; Pathogenesis of Various Forms of Choroidal Tuberculosis: Rôle of Superinfection, Dr. H. Lagrange, Paris.

Moorfields Hospital.—In the annual report of the Royal London Ophthalmic Hospital (Moorfields) for 1930, it is announced that the first portion of the program of enlargement and reconstruction has been completed at a cost of 41,365 pounds. A new private ward and two additional blocks for the nursing and domestic staff were opened last December. It is hoped during the present year to extend the existing outpatient department, and in the building thus erected to provide additional accommodation for the medical school. The number of available beds has been increased to 152. Last year 52,372 new outpatients were treated, a larger number than in either of the two previous years. There is a surplus of 1,798 pounds on the year's working, and 13,500 pounds is available toward the second portion of the extension scheme, which will cost about 40,000 pounds. The draft plans for this are already under consideration. (From the *British Medical Journal*, Aug. 29, 1931.)

DEATHS

Prof. K. K. Lundsgaard of Copenhagen, Denmark, died on August 30, of pneumonia. Dr. Lundsgaard was professor of ophthalmology in the University of Copenhagen, president of the next International Congress of Ophthalmology to be held at Madrid in 1933 and editor of the *Acta ophthalmologica*. A full obituary will appear later.

Obituary

LAWRENCE WEBSTER FOX, M.D.

1853-1931

In the death of Dr. Lawrence Webster Fox on June 4, 1931, at Ardmore, Pa., after a distressing illness induced by a cardiorenal disease of nearly a year's duration, the Graduate School of the University of Pennsylvania and a large, distinguished and widespread clientele have lost a notable authority in the science and art of ophthalmology.

At the time of his death, Dr. Fox was professor of ophthalmology and a member of the board of managers of the Graduate School of the University. He was born on March 19, 1853, at Hummelstown, Pa., the son of the late Dr. Thomas G. Fox. He was educated in the public schools of his native town and at the Millersville State Normal School, from which he graduated in 1872. Following his graduation, he became associated in his twenty-first year with the well known firm of John E. Fox and Company, bankers and brokers of Philadelphia. Deciding to embark, a few years later, on a medical career, he entered Jefferson Medical College, from which institution he graduated in 1878.

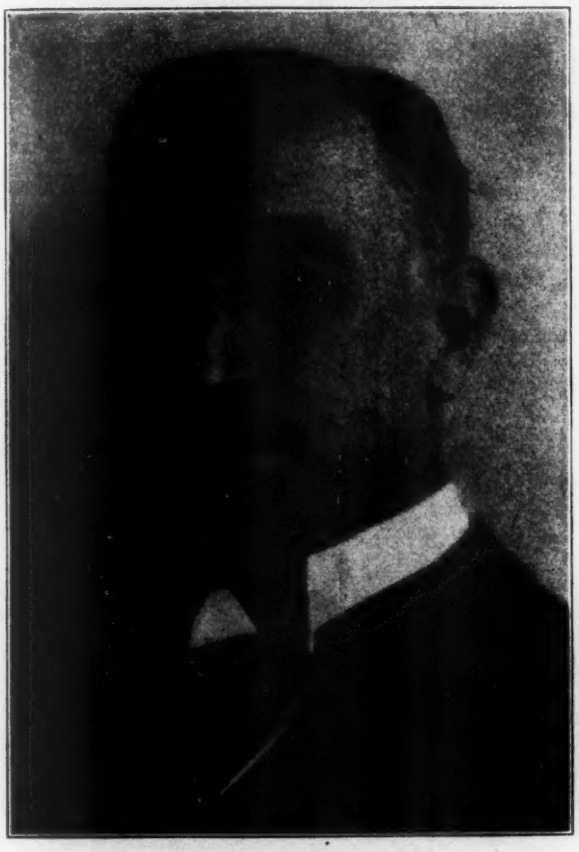
His medical education in ophthalmology was continued abroad at the University of Vienna under Professors Mauthner and Ernest Fuchs. A class of ten American students was organized by Dr. Fox for study under Dr. Fuchs, which marked the beginning of a friendship with the great master in ophthalmology, which remained unbroken until his death.

Dr. Fox subsequently spent two years at Moorfields Eye Hospital in London. On his return to America he located in Philadelphia and entered actively on the practice of ophthalmology. At this period he became an assistant in the department of eye diseases of Jefferson Medical College under Prof. William Thompson, and subsequently, chief of the clinic. On the organization of the Medico-Chirurgical College in 1893, he became professor of ophthalmology, and after the merger of that institution with the University of Pennsylvania, he continued as professor of ophthalmology in the Medico-Chirurgical College Graduate School of Medicine until his death.

Dr. Fox was a co-author of a quiz compend on the eye, the first and second editions of which appeared in 1886 and 1888, respectively. He was the author of a standard treatise on diseases of the eye published in 1904, which was favorably received and which passed through four editions. He published also a considerable group of articles dealing mainly with clinical ophthalmology.

Dr. Fox will be especially remembered as a bold and brilliant operator. Indeed, there were few important types of operative procedure that one could not see illustrated in a series of visits to his clinical amphitheater. The memory of a subtle and finished technic will linger with those who had the opportunity of such an association.

For many years he was especially interested in the operative treatment for trachoma, which found expression in much clinical work in



LAWRENCE WEBSTER FOX, M.D.
1853-1931

grattage and tarsectomy at his clinic, as well as in his visitation to a number of the Indian Reservations.

Among the important public positions held by Dr. Fox were: president of the board of trustees of the Pennsylvania Military Academy at Chester, Pa.; member of the State Council of the Blind; member of the board of managers of the Orthopedic Hospital of Philadelphia and president of the board of directors of the Pennsylvania Home Teaching Society and Free Circulating Library for the Blind.

Dr. Fox married Miss Bickerton of Liverpool, England, daughter of Mr. Thomas Bickerton and sister of Mr. Herbert Bickerton, both father and brother being distinguished authorities in ophthalmology. Two children resulted from this union, a son, Lawrence Webster, Jr., and a daughter, Beatrice, now Mrs. Griffith, who is a sculptress of notable ability. The widow and the two children survive him.

Striking in physique, kindly and unassuming in manner, untiring in his devotion to his professional work, Dr. Fox attained a distinguished place in American ophthalmology that will be exceedingly difficult to fill.

G. Oram Ring, M.D.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Bacteriology and Serology

BACTERIOLOGIC OBSERVATIONS IN PREXEROSIS CORNEAE. A. PILLAT, Arch. f. Ophth. **125: 173**, 1930.

In a previous paper (*Arch. f. Ophth.* **124:** 486, 1930), Pilat described a new clinical entity, the prexerosis corneae in adults, caused by lack of vitamin A in the food. Smears taken from the eyes of these patients show always characteristic bacteriologic flora, namely, arranged according to their relative frequencies, xerosis bacilli, pneumococci, staphylococci and Koch-Weeks bacilli. All these germs are to be considered as saprophytes, which find a good medium in the degenerated epithelial cells. They are found on the cornea and on every part of the conjunctiva. The bacteriologic findings are so definite and constant that it seems justified to make the diagnosis prexerosis from the smear alone, even if the clinical symptoms (Bitot spots, hemeralopia and wrinkling of the conjunctiva bulbi) are not present. If the prexerosis is combined with a phlyctenular keratoconjunctivitis or any other acute inflammation, the growth of the saprophytes is inhibited. Smears taken from places around phlyctenules are frequently found free from bacteria.

P. C. KRONFELD.

Conjunctiva

SURGICAL TREATMENT OF PTERYGIUM WITH MUCOUS AUTOPLASTY. PEDRO MATA, Arch. de oftal. hispano-am. **31: 177** (March) 1931.

The procedure advocated by the author is that of Duverger and Velter. After preliminary anesthesia, he removes the head of the pterygium with a Graefe knife after transfixion; he next removes the conjunctival portion with scissors; he then circumscribes and excises a semilunar flap from the mucous membrane of the lower lip, which he transplants to the conjunctival defect, fixing it there by means of sutures. He reports on thirty-four cases.

C. E. FINLAY.

LYMPH NODES IN THE CONJUNCTIVA OF MAMMALS. K. HIWATARI, Arch. f. Ophth. **125: 403**, 1930.

The normal conjunctiva of dogs and rabbits looks perfectly smooth macroscopically and is free from lymph nodes on histologic examination. Any sort of irritation leads to a formation of lymph nodes which, if the irritation ceases, disappear rapidly. Lymph nodes in the conjunctiva always indicate a pathologic process.

P. C. KRONFELD.

ANKYLOSING TUBERCULOSIS OF CONJUNCTIVA ASSOCIATED WITH BILATERAL EXUBERANT CYSTS OF KRAUSE'S GLANDS AND CYSTIC DEGENERATION OF THE MEIBOMIAN GLANDS. D. MICHAÏL, Cluj. med. 11: 273 (June 1) 1930.

A woman, 26 years of age, suffered from ulcerovegetative tuberculosis of the conjunctiva tarsi and the inferior transition fold. Excisions of the diseased portions entailed improvement of the subjective and objective condition, but there occurred in the course of three years cicatricial retractions in the conjunctival sac, vascularization and opacities of the cornea, and impairment of the acuity of vision. At the end of four years there was complete symblepharon; the acuity of vision was about perception of hand movements, and an ovoid, tumor-like mass was palpable in the upper eyelid. Operation proved this tumor to be a cyst. The histologic examination of the whole of the excised tarsus showed evidence of a lymphoid-epithelioid infiltration with giant cells. In the pretarsal tissue there was a lymphatic infiltration by lymphocytes, plasma cells, epithelioid cells and giant cells. There was also found the wall of the cyst, consisting of epithelial cells. The tarsal tissue showed the maximum of the tuberculous lesions on the ciliary margin. The meibomian glands were completely destroyed, and their excretory ducts were enormously dilated and transformed into cystic cavities. In the supratarsal tissue the accessory lacrimal glands of Krause revealed a lymphatic infiltration and dilated lumina simulating cysts. So one had in this case to deal with a severe, progressive bilateral tuberculosis of the conjunctiva resulting in ankyloblepharon and xerosis. The cystic degenerations of the meibomian glands and Krause's glands make this case interesting from the pathologic point of view, as such an outcome is commonly enough to be met with in trachoma and pemphigus, but not in tuberculosis. The cysts of the meibomian glands were due to a meibomian pericanaliculitis juxta-orificialis. The large cyst of Krause's glands, a clinical rarity, too, arose in the same manner. N. BLATT.

Congenital Anomalies

MULTIPLE AND BILATERAL CONGENITAL MALDEVELOPMENTS IN A PATIENT WITH SPECIFIC CHORIORETINITIS. NICOLAU-CLIMESCU, Cluj. med. 11: 420, 1930.

A man, 47 years of age, had undergone a marked impairment of vision in a few months. He had bilateral choroiditis with vision as follows: right eye, finger counting at 1 meter; left eye, finger counting at 2 meters. He presented a series of malformations as follows: slight protrusion of the disks and irregularity of their borders, dimples on the disk, and an epipapillary membrane in the right eye. After two years, vision was: right eye, finger counting at 2 meters; left eye, finger counting at 4.5 meters. In the right eye, the visual field for white and colors was markedly contracted.

The authors are of the opinion that the functional troubles were due to the specific chorioretinitic process, but not to the congenital malformations. N. BLATT.

Cornea and Sclera

KAYSER-FLEISCHER RING IN THE CORNEA IN A CASE OF WILSON'S DISEASE. D. L. POE, Am. J. Ophth. 13: 1045 (Dec.) 1930.

A man, aged 21, began having rheumatic pains and weakness two or three years before observation. Examination showed a typical facies, muffled speech, a coarse rhythmic tremor of all extremities but especially the right upper, a pill-rolling position of the fingers, a somewhat stiff carriage, sluggish pupils, a few nystagmoid jerks in the horizontal and vertical planes, poor convergence, a moist tongue and mouth and inability to close the mouth well.

The cornea presented a circular brownish-green pigmentation which extended all around the limbus. It resembled the Kayser-Fleischer ring of Westphal-Strümpell pseudosclerosis or Wilson's hepatolenticular degeneration. Slit-lamp study showed this pigmentation to be in Descemet's membrane. It was 2.5 mm. broad, and no clear corneal substance was seen between it and the limbus. This was confirmed by gonioscopic examination, which showed the ring to be confined to the cornea. No sunflower lens opacity could be found. The author discusses the possible cause of Wilson's disease and the manner in which the cornea is involved. His conclusions are: "1. The object of this paper is to call attention to the characteristics of a Kayser-Fleischer ring. 2. It is regarded by many authorities as a pathognomonic sign in hepatolenticular degeneration. 3. Because of the similarity of a large number of clinical symptoms found in this disease and in chronic encephalitis, the former is frequently mistaken for the latter ailment. 4. A degenerative process of the liver exists in Wilson's disease and does not necessarily exist in chronic encephalitis, but as yet we have been unable clinically to establish the presence of a cirrhotic liver with any degree of certainty; therefore we must rely to a great extent upon the presence of a Kayser-Fleischer ring for a differential diagnosis. 5. Hepatolenticular degeneration is usually progressive and fatal. The span of life after the disease becomes established ranges from two to seven years, while in chronic encephalitis it is not necessarily progressive, or fatal, in the same period of time."

W. S. REESE.

ARGYROSIS CORNEAE. B. FRIEDMAN and A. RÖTTH, Am. J. Ophth. 13: 1050 (Dec.) 1930.

The first slit-lamp picture of argyrosis was published in 1927 by Larsen. The authors examined twelve trachomatous patients with argyrosis, and in all but two a distinct golden to greenish coloration of the posterior corneal surface was observed. In some cases there was a similar golden reflex from the anterior capsule. In others the posterior capsule and the subcapsular part of the posterior cortex were yellowish. Two illustrative cases are reported.

The authors discuss corneal pigmentation following exposure to other metals. They remark on the similarity of argyrosis corneae to the golden ring found in pseudosclerosis.

The following summary is given: "In ten out of twelve cases of conjunctival argyrosis a corneal argyrosis was seen with the slit-lamp.

Even moderate conjunctival argyrosis was accompanied by corneal argyrosis. This condition is strongest at the limbus and diminishes toward the center and again becomes a little more pronounced at the upper limbus. The color variation between the strongest and the weakest cases may be a golden brown, yellowish gold, yellowish green, greenish blue or ultramarine. The pigment layer appears in most cases to be finely stippled and lies at the posterior surface of the optical section, probably between Descemet's membrane and the endothelium. In more pronounced cases it piles up forwards. There is in some cases also an argyrosis of the lens. It appears as a yellowish reflex from the anterior capsule, and as a diffuse yellowish coloring of the posterior capsule and the outermost layers of the posterior cortex. The scleral conjunctiva is tinged with gold in nearly all cases."

W. S. REESE.

BIOMICROSCOPIC STUDY OF LEPROUS KERATITIS. ANTONIO MORATAL, Arch. de oftal. hispano-am. **31**: 234 (April) 1931.

This study is based on a biomicroscopic study of the cornea in one hundred lepers from the Fontilles asylum. Eighty-four per cent of these showed corneal lesions; 50 per cent of those in the first five years of their infection had lesions of the cornea; of those whose infection had been present between five and ten years only two had healthy corneas. The corneal lesions were primary or secondary to iritis or to scleritis. He distinguishes four varieties of corneal lesions: (1) keratitis punctata, (2) hyperplastic keratitis, (3) corneal ulcerations (mostly complications) and (4) sector-like infiltration.

C. E. FINLAY.

Experimental Pathology

CHANGES IN THE LENS DUE TO ULTRAVIOLET RAYS. P. SCOTTI, Ann. di ottal. e clin. ocul. **58**: 963 (Nov.) 1930; **59**: 19 (Jan.); 230 (March) 1931.

The author reviews the literature on the effect of ultraviolet irradiation on the lens. By the histologic study of eyes with and without a lens, which had been exposed to ultraviolet rays, and by spectrophotographs taken through a layer of lens tissue, it seems definitely established that the lens absorbs much of the light below 4,000 Angström units—25 per cent of all the ultraviolet rays, according to Duke-Elder. Glassblowers' cataract, once considered due to ultraviolet rays is now known to be caused by heat rays. The results of various attempts to produce cataract by ultraviolet irradiation are reviewed.

The author irradiated solutions of egg albumin and extracts of lens in physiologic solution of sodium chloride with the mercury vapor arc. These solutions coagulated after exposures of from two to six hours even when a filter of glass cut off the ultraviolet rays. Extracted beef lenses developed opacities after from three to seven hours of irradiation; these were most marked in the nuclear region and were

seen only where no glass filter was employed. The same changes were seen when whole enucleated eyes were irradiated through the cornea.

The eyes of living rabbits were irradiated with the same lamp for one-half hour to fifty hours, and the lenses were examined in focal light and in sections. The longer exposures were divided into various sittings. In these animals exposure for longer than three hours caused the formation of vacuoles in the capsular epithelium. Longer exposures caused an increase in the visibility of the suture lines, especially near the posterior pole, an increased refraction of the nucleus and the appearance of vacuoles in the cortex. These phenomena disappeared within a day or two after irradiation. After still longer exposures, true opacities appeared in the cortex due to swelling of the fibers and the accumulation of fluid between the fibers and of amorphous material replacing some of them. These changes were seen just beneath the capsule. These opacities sometimes formed several days after the last irradiation, and remained unchanged during periods of observation up to two months.

Edema and congestion of the ciliary body and stratification of its epithelium were seen only after the longer exposures, longer than were required to produce changes in the lens capsule, for which only from three to eight hours were necessary. In a few eyes removed some time after the last exposure, proliferation of the capsular epithelium could be seen. For short circuit cataract the author believes that ultraviolet rays can hardly be held responsible, since it has been caused by a very short exposure to light containing sometimes only small amounts of ultraviolet rays but large amounts of heat rays, while his experiments showed that exposures to intense ultraviolet rays for less than three hours produced no changes in the lens. He believes that the factor of ultraviolet irradiation can also be excluded in glassblowers' cataract.

In considering the pathogenesis of the lens changes caused by ultraviolet rays, the author believes that the effect on the ciliary body can be excluded, since changes here were often entirely absent where lens changes had occurred. Changes in the capsular epithelium are more important, since the cortical opacities never occurred without such changes in the capsule and were always in the part of the lens where such changes were most marked. The effect of such changes on the lens fibers is probably through a disturbance of the processes of osmosis on which the lens depends for its nutrition, and only in the most advanced stages is due to increased absorption of water through the injured capsule. The capsular changes themselves are apparently due to a direct coagulating effect of the ultraviolet rays on the protoplasm of the epithelial cells. True opacities of the lens were formed only after twelve hours of continuous irradiation or twenty-four hours irradiation in divided doses. This and other facts lead the author to discard the factor of ultraviolet irradiation in the production of senile cataracts.

A bibliography and twenty-five photographs and photomicrographs are included.

S. R. GIFFORD.

THE EXPERIMENTAL X-RAY AND RADIUM CATARACT. L. PETER, Arch. f. Ophth. **125:** 428, 1930.

In rabbits 1 erythema dose of x-rays can produce permanent lenticular lesions of the type of a posterior axial complicated cataract. This opacity becomes visible 165 days after the exposure. Therefore one has to wait at least five months before a certain dose of x-rays can be said to be harmless for the lens. The same holds true for radium with the usual 0.5 mm. platinum iridium filter. The smallest dose used was 144 mg. element hours, which after five and one-third months produced definite opacities in the lens. Axial parts of the cortex which contain no nuclei seem to be more sensitive to x-rays and radium than any other part of the lens. The epithelium of the lens does not seem to be affected by the small doses of x-ray. It is probable that the rays act on the living fibers of the lens, which are next to be sclerosed. These fibers become opaque and are later on crowded inward by healthy fibers formed afterward. Yet it occurs sometimes that opaque parts remain for a long time immediately underneath the capsule. The x-ray and radium cataract differs from the infra-red cataract in its long period of latency.

P. C. KRONFELD.

General Diseases

ARACHNODACTYLIA. H. WEVE, Arch. f. Augenh. **104:** 1 (Jan.) 1931.

The author gives a sketch of arachnodactylia or dolichostenomelia based on a series of his own cases as well as a review of the literature. He reports twenty-three cases from six families. These are added to the sixty cases already reported in the literature. The clinical entity is apparently more common in Holland than the literature would lead one to believe. Women are not more frequently affected than men. The disease is essentially familial and this becomes especially apparent if one includes the arrested cases, so-called formes frustes.

It is an inherited character and essentially dominant. About half of the children of one family are affected, but usually only a single branch of a large family. The cause of this incomplete dominant mode of inheritance cannot be accounted for. Basal metabolic studies made on these cases suggest that the syndrome is a congenital form of mesodermal dystrophy. The classic picture of the disease consists essentially in an abnormal length and thinness of the extremities, especially the hands and feet. In the well marked cases all the bones of the arm are abnormally long and the abnormal proportions seem to increase from the shoulder to the fingers; in other words, the disease can be most easily detected by a glance at the hands. The muscles show no paralyses or appearance of degeneration. Occasionally contractures of the joints are met with, from which the disease gets its name, the fingers becoming like spider feet. In from 40 to 50 per cent of these cases there are bilateral congenital ocular anomalies. For the most part these consist in luxation or subluxation of the lens. As a consequence of the more spherical form of the subluxated lens there may develop a high myopia, or, if the edge of the lens lies below the pupil opening, a marked hypermetropia may be present. F. H. ADLER.

Glaucoma

POSTERIOR SCLERECTOMY IN THE TREATMENT OF GLAUCOMA DUE TO TOTAL POSTERIOR SYNECHIAE. A. NICOLATO, Arch. di ottal. **38:** 162, 1931.

The author calls attention to the group of cases in which the iris is bound down to the lens over the greater part of its posterior surface, so that iris puncture, as practiced for iris bombé, is impossible, and iridectomy is likely to prove unsuccessful, besides involving the danger of a new attack of iridocyclitis. He calls this condition "iris-block." Simple posterior sclerotomy gives relief from tension, but its effect is usually only temporary. Hence the author was led to trephine the sclera with a 3 to 4 mm. trephine, usually between the superior and external recti. The scleral button is removed without injuring the choroid, and then the choroid and retina are punctured with a Graefe knife, allowing the escape of a small amount of vitreous. The previously prepared conjunctival flap is then sutured.

Four cases are reported of painful eyes, blind or practically so, with a tension of 84, 96, 84 and 96 mm. (Schiötz), respectively. In all cases the tension was reduced to normal or nearly so, following this procedure; pain and congestion disappeared, and the eye was retained. The author believes that the reduction of tension by this method is permanent, but his cases were not followed for periods longer than one or two months.

S. R. GIFFORD.

THE GLAUCOMA PROBLEM. E. P. FORTIN, Arch. de oftal. de Buenos Aires **6:** 219 (April) 1931.

The author refers to the improvement produced in glaucoma by forced exercise of accommodation: making the patient read fine print such as logarithm tables, a method made use of by ophthalmologists prior to the employment of iridectomy and myotics. The benefit derived is due to a contraction of the ciliary muscle and pupil with the opening of Schlemm's canal and enlargement of the spaces of the pectineal filter and of the venous and lymph spaces at the corneal periphery, putting the uveal fluid into motion. These views are supported by a painstaking anatomic study and beautifully illustrated by photomicrographs.

C. E. FINLAY.

Injuries

INTRA-OCULAR INJURY AND EXTRA-OCULAR FOREIGN BODIES IN THE ORBIT. VAN DER STRAETEN, Arch. d'opht. **48:** 34 (Jan.) 1931.

The author reports three cases of foreign body of the orbit because he thinks that they illustrate the following facts: 1. Intra-ocular lesions resulting from the projection of a foreign body into the orbit can lead to the belief that the foreign body is within the globe when there has been no penetration. 2. Penetrating wounds of the eye can lead to the belief that the foreign body producing them is in the eye, though it has completely traversed the eye and is lodged in the tissues of the

orbit. 3. Although clinical observation over a period of time will result in the proper diagnosis, roentgenographic examination according to an approved technic quickly and surely settles the question. 4. The use of Wessely's shell is a simple and efficient method of localization. 5. Movement of the foreign body with the eye does not definitely prove it to be within the eye. 6. Contusion can produce intra-ocular lesions at the site of injury.

The author urges the use of a shell for localization, believing that it is the simplest procedure available.

S. B. MARLOW.

PRACTICAL OBSERVATIONS ON THE PATHOLOGY AND THERAPY OF HYDROGEN SULPHIDE INFLAMMATION OF THE EYES. F. STOEKER, Med. Klin. **27:** 16 (Jan. 30) 1931.

Workers in artificial silk and like commodities are exposed to fumes of hydrogen sulphide. With improvement in methods of ventilation these cases are not so numerous or so severe. Patients with mild cases are treated at the plant, and only severe cases are seen by the ophthalmologist. The lesion consists of punctate or confluent epithelial clouds in the inner layers of the cornea. Signs of irritation of the conjunctiva with epiphora and pain are noted. The instillation of cocaine and epinephrine has been the usual procedure to relieve the pain. A nonirritating salve of petrolatum and hydrous wool fat aids healing. Recently the Ciba Company has brought out *di*-allyloxyethyl-*di*-phenyl, which does not burn or cause clouding of the cornea or dilatation of the pupil as cocaine does. A 2 per cent solution or an ointment of *di*-allyloxyethyl-*di*-phenyl, epinephrine or ephedrine in petrolatum gives excellent results. The author recommends isotonic saline or boric acid solution together with an ointment of petrolatum or olive oil with hydrous wool fat for mild cases. Jaensch's work on this condition draws comment.

L. L. MAYER.

Lens

HISTORICAL AND CRITICAL EVALUATION OF THE RING-SHAPED OPACITY OF THE ANTERIOR CAPSULE OF THE LENS AFTER CONTUSIONS. M. HANDMANN, Klin. Monatsbl. f. Augenh. (Beilageheft) **85:** 79 (Dec.) 1930.

Vossius described the ring-shaped opacity resulting from contusions as an imprint of the inner surface of the iris on the anterior capsule. Handmann, after studying and observing eight cases in his own practice, rejects this opinion. He supports his views by quoting publications on the subject. Contusions, in his opinion, are the most frequent but not the sole causes of this phenomenon. Spontaneous hemorrhages and hemorrhages occurring at long intervals after the contusions are named among the causes. The ring-shaped opacity cannot be caused by pressure of the pupillary margin against the anterior capsule. It is actually a ring-shaped or disk-shaped deposit derived from the aqueous humor and located on and in the anterior capsule. This opacity, measuring usually from 3 to 4 mm. in diameter, can develop because the

pupillary portion of the anterior capsule is predisposed to such a formation, owing to its roughness, porosity and osmosis. Temporary interruption or retardation of the current in the anterior chamber may further the development of the ring or disk. The origin of the floccular deposits on the pupillary margin, described by Kraupa, Vogt and Handmann, are related to the phenomenon under discussion. The rarity of the ring-shaped or disk-shaped opacity is explained by individual differences in the fine structures of the anterior capsule. It can develop only if a number of favorable factors are present. The peripheral zone of the anterior capsule is never involved. Uncertainty still exists regarding the morphologic nature of the deposits. They may consist of pigment or of blood and complicated derivations of blood.

K. L. STOLL.

INTRACAPSULAR EXTRACTION OF SENILE CATARACTS IN A CASE OF KERATOCONUS. A. ELSCHNIG, Klin. Monatsbl. f. Augenh. **86:** 79 (Jan.) 1931.

The author knows of only three cases of keratoconus in which cataracts were extracted: one of Schnabel's and two of Axenfeld's. The case reported here by Elsching is that of a healthy and robust man, aged 64, whose right eye had been weak since childhood, while the left was myopic. Faint opacities could be seen with the slit-lamp at the apex of the right keratoconus. The lens presented a uniform brownish opacification; fingers could be counted at a distance of 1 meter. The keratoconus of the left eye was of a minor degree and free from opacities; vision was 1/36 with about 4 concave diopters. Intracapsular extractions with a round pupil were done on both eyes. The course of recovery was normal, and the result satisfactory. In the right eye, with + 6.0 diopters sphere ⊃ + 5.0 cylinder, axis 180, vision was 6/24; in the left eye, with + 9.0 diopters sphere ⊃ + 3.0 cylinder, axis 170, 6/8. Nieden type 1 could be read with the proper addition for close range. The keratoconus of both eyes had been stationary for many years.

K. L. STOLL.

SENILE CATARACT, LATENT SYPHILIS AND POSTOPERATIVE PLASTIC IRITIS. E. PUSCARIU, Cluj. med. **11:** 418, 1930.

In a previous paper Puscariu has recorded evidence that many cases of postoperative plastic iritis concomitant with cataract are caused by latent syphilis. The serum reaction test for syphilis resulted positively in 60 per cent of such cases. These cases of iritis had never been associated with cyclitis, and the antisyphilitic treatment—carried out at the same time as the local treatment—led, for the most part, also to improvement of the acuity of vision. Two such cases are reviewed. The first patient was operated on for a cataract of the right eye. The serum reaction was positive for syphilis. After five days there appeared a plastic iritis, which improved owing to local treatment and injections of mercury. She left the clinic with vision equaling finger counting at 1 meter. The antisyphilitic treatment was continued. After one year the left eye was operated on for cataract. This time there was no iritis. Vision was: right eye, 0.5; left eye, 0.7. The second case ran the same course. Puscariu feels that in postoperative iritides a conspicuous part must be ascribed to latent syphilis, this part being at any rate greater

than that of the toxic action of the remaining cortical and capsular fragments so often made responsible for the genesis of postoperative iritides. Besides, in the two cases presented, no lenticular remnants could be evidenced.

N. BLATT.

Neurology

PROOFS OF THE HYPOPHYSEAL MECHANISM OF OPHTHALMIC MIGRAINE. J. SÉDILLOT, Clinique, Paris **25:** 113 (March) 1930.

The paper is based on anatomy and on clinical observations. There is a difference in so-called "common" migraine and ophthalmic migraine. The latter comes on suddenly, at any hour of the day, and is accompanied by visual disturbances especially noticeable at the onset. The former is vague and more lasting. Theories as to the mechanism of ophthalmic migraine are many. The two successive ocular phenomena noted at the beginning of the attack, that is, scintillating scotomas, symptomatic of nerve irritation, and lateral homonymus hemianopia, symptomatic of inhibition in the nerve, are due to compression of the optic tract by the hypophysis. Anatomically the tract is wedged between the internal carotid and the hypophysis. In the initial stage there is evidence of irritation due to compression, later comes the interference with the conducting pathway. The author feels that all symptoms may be explained by this hypothesis.

L. L. MAYER.

TREATMENT OF SCOTOMA SCINTILLANS. J. ARRUGA, Arch. de oftal. hispano-am. **31:** 103 (Feb.) 1931.

After brief remarks on the nature of the trouble, the author recommends the employment of phenobarbital, as first advised by Lindmeyer in 1927, used in daily doses of from 0.025 to 0.1 Gm. during the intervals, combined with regulation of the stools, sleep, diet and work and avoidance of causes of worry and excessive fatigue.

C. E. FINLAY.

TUMORS OF THE HYPOPHYSIS AND THEIR TREATMENT. O. HIRSCH, Klin. Monatsbl. f. Augenh. **85:** 609 (Nov. 28) 1930.

In the introduction the author gives a short survey of the various types of tumors of the hypophysis from both the pathologico-anatomic and the surgical standpoints. Disturbances of central vision and of the field of vision and changes of the optic nerve were found in all cases, except in those of acromegaly. The latter are characterized by the absence of visual disorder. Neuritis and, frequently, choked disk were observed in suprasellar tumors of the pedicle of the hypophysis. The defects in the field of vision were not characteristic. Atrophy of the optic nerve was present in suprasellar meningiomas and intracranial complications; bitemporal hemianopia or central scotomas were recorded in the initial stage of the latter. No visual disturbances were seen in the intrasellar tumors. Vision varied between normal in one eye and diminution in the other, to amaurosis in one eye and failing vision in the other. Temporal and bitemporal hemianopia were found in 84 per cent of the cases. This symptom might be considered typical of tumors of the hypophysis, were it not met with occasionally in locomotor ataxia and in internal hydrocephalus. A number of cases pre-

sented differences between both eyes: temporal hemianopia for colors in one eye with a normal field for white in both eyes and a normal color field in the other eye; or temporal hemianopia for white and colors in one eye only, or coupled with preservation of the upper nasal quadrant of the second eye. Bitemporal hemianopia for colors with a normal field for white existed in one case in which a central scotoma for white was found in one eye; the temporal hemianopia was complete for white and colors in other cases. Homonymous hemianopia was present in 7 per cent of the author's cases; defects of the nasal field of one eye, in 4.4 per cent. Central scotomas, observed in the initial stage of 11 per cent of the cases were at first wrongly interpreted as symptoms of retrobulbar neuritis or multiple sclerosis. Primary atrophy of the optic nerve was seen in 89 per cent of the cases, optic neuritis in 4.4 per cent and choked disk in 8.9 per cent. The latter two categories are so rare that they can be used for the diagnosis solely in connection with other symptoms and with an affirmative roentgenogram. The disk was normal, but the vision was greatly reduced in some cases, while normal vision was coupled with pale disks in others, a condition not identical with degeneration of the optic nerve fibers in tumors of the hypophysis, according to Walker and Cushing.

Shadows of lime concretions in and above the region of the sella on the roentgenogram are often indicative of cystic tumors of the hypophysis. A greatly enlarged sella, on the other hand, is not characteristic of a tumor of the hypophysis; it may be found also in hydrocephalus and several forms of tumors of the brain, complicated by hydrocephalus. Choked disk and the picture of an enlarged sella can be used for the diagnosis of a tumor of the hypophysis only in connection with other typical symptoms. In their absence Sgallitzer's method of filling the spinal canal with iodized poppy seed oil 40 per cent may be used to advantage. The iodized oil ascends into the third ventricle and forms a curved line high above the sella in tumors of the hypophysis, while only droplets of the fluid are seen in the area of the sella in hydrocephalus. A normal sella is not indicative of the absence of a tumor of the hypophysis. Suprasellar tumors and meningiomas of the chiasmatic sulcus occur without an enlarged sella.

Four operative methods are described, those of Krause and Schlosser, Cushing's sublabial-septal method and Hirsch's method. Hirsch abandoned his ethmoidal method. He now practices the septal method which is described in detail. A submucous resection of the cartilage of the septum is made as far back as to the anterior wall of the sphenoid cavity. The space obtained after the removal of the cartilage will allow for the opening of the sphenoid, the elimination of the rostrum and the removal of the anterior walls of the sphenoid. The enlarged sella is chiseled open and its bony portion removed. The tumor is removed with curets. Solid tumors receive postoperative treatment with radium, by means of an appliance devised by the author. Hirsch operated on 161 patients, between 1910 and 1929, with 14 deaths following the operation (8.7 per cent) and 4 deaths after secondary operations for recurrences, or a total loss of 10 per cent. The mortality in 103 patients, operated on between 1919 and 1929, was 5 after the operation, then 12 within three years (8 of these inoperable recurrences) and 4 within

from three to six years later; 2 of these patients, also with inoperable recurrences, died after six years. Of 29 patients operated on from four to ten years before the report, 22 obtained a permanent result; one case remained stationary, one patient suffered a recurrence after an initially good result and in one the operation was unsuccessful; no information could be obtained from 4 other patients. Hirsch added charts of the visual fields of 7 of his earliest patients operated on from six to eleven years before. These illustrate the condition before the operation as well as from ten to eleven years later. Hemianopia for white and colors had disappeared, and the greater portions of the fields had returned. Vision had increased considerably; in some cases it had even become normal.

K. L. STOLL.

Ocular Muscles

LATENT HETEROPHORIA AS NOTED AFTER PROLONGED MONOCULAR OCCLUSION. C. M. SWAB, Am. J. Ophth. **13**: 1054 (Dec.) 1930.

The author has used occlusion tests since 1924. He considers short occlusion unsatisfactory, and in all the cases of this series (amounting to well over 100) occlusion was employed for not less than forty-eight hours. A dry sterile pad was used and sufficient adhesive to exclude all light. When this was removed, a definite routine was followed. The author recommends occlusion in the following instances: 1. Patients who are not relieved of asthenopic symptoms when provided with lenses that correct the ametropia. 2. Persons without demonstrable ocular disease, who are exceptionally sensitive to light. 3. Dissociation in the muscle balance between the findings at 6 meters and at 33 cm.; e. g., right hyperphoria for the one, and left hyperphoria for the other. 4. Persons who state that they cannot read for more than thirty minutes at one time. 5. Any so-called neurasthenic patient with asthenopia. 6. Patients who have "panoramic" headaches, vertigo and nausea which come on while riding on moving vehicles or while watching the cinema. 7. Persons who are wearing prisms that suit their needs according to manifest tests but who still complain of asthenopia, as discussed by Beach. 8. Persons who have quivering or twitching of the lids from intermittent spasm of fibers of the orbicularis oculi.

Four illustrative cases are reported, and the following conclusions drawn: Prolonged monocular occlusion is a valuable measure when evaluated in its true perspective. It is not meant to replace other measures in routine examination. Its use is called for in the unusual cases of asthenopia which do not respond to the accepted treatment.

W. S. REESE.

OCULOMOTOR PALSY WITH CYCLIC INNERVATION OF THE INNER EYE MUSCLES. R. STEIN, Med. Klin. **27**: 350 (March 6) 1931.

This is the report of a case of so-called "congenital cyclic oculomotor disease," which was first described by Axenfeld and Schürenberg in 1901. About thirty-two cases of this special type of oculomotor palsy have been reported in the literature.

The patient, aged 25, had had a severe fever when about 6 months old, and his eyes crossed at that time. Examination gave negative results except for the eye muscle defects. The right eye deviated outward 15 degrees and upward 8 degrees on fixation with the left eye. Adduction past the midfixation line was absent. Downward motion was limited to the action of the superior oblique. Upward movement was limited to about 2 mm., and on closure of the lid a modified Bell's phenomenon—outward movement of the globe—was elicited. A paralysis of the levator existed which caused the right palpebral fissure to be only from 3 to 4 mm. wide. This was increased only from 1 to 2 mm. on effort, but on adduction it widened to 8 or 9 mm. The pupil was sluggish to both direct and indirect light stimuli; convergence was absent and accommodation affected. The lid closure reaction was diminished, and stimulation was without effect. The cyclic phenomena consisted in a sort of hippus of the pupil which varied from 2 or 3 to 7 or 8 mm. in from ten to twenty seconds. These spasms occurred at from one to two minute intervals, and there was an associated spasm of the ciliary muscle which changed the refraction and the vision. An operation to correct the strabismus had no effect on these phenomena. The various theories as to localization of the lesion and the cause of the automatic rhythm are reviewed. The author believes that only an anatomic study of such a case will answer the many questions. A bibliography is appended.

L. L. MAYER.

Pharmacology

THE ACTION OF "HARMINE" ON THE EYE. G. SALVA, Arch. di ottal. 38: 197, 1931.

The effects of "harmine," which has recently been used in treating patients for the symptoms of parkinsonism, on the eye were investigated. It is an alkaloid derived from *Peganum harmala,* which grows in Asia and North Africa and which is almost identical with a South American plant from which banisterine is derived.

A 1 per cent solution of the hydrochloride was instilled into the conjunctival sac of the author and other subjects. A burning sensation was felt for several seconds, followed by anesthesia which began in five minutes, was complete after six or seven minutes and lasted from twenty to thirty minutes. Edema of the corneal epithelium, manifesting itself by fine punctate opacities and microscopic bullae, was produced. The conjunctival capillaries were dilated, and the conjunctiva showed slight chemosis which disappeared after one hour. Vision, accommodation and the size of the pupil were unaffected, and no change in intraocular tension was produced. No toxic effects suggesting individual intolerance to the drug were observed.

S. R. GIFFORD.

The Pupil

THE SHARE OF EACH MUSCLE OF THE IRIS IN THE PUPILLARY REACTIONS. C. BEHR, Arch. f. Ophth. 125: 147, 1930.

This paper is based on clinical observation of a patient, 25 years of age, whose one eye, following a contusion, showed a separation of the

stroma of the iris from the pigment layer throughout one tenth of its circumference. The two layers could be seen to contract or relax independently of each other. In this eye it was found that the reflex of the pupil to light was produced by an increased tone of the sphincter plus active relaxation of the dilator.

The same mechanism was observed when the pupil reacted to accommodation. Throughout the course of the psychic reaction of the pupil, the dilator, the tone of which was actively increased, was the predominating muscle, and relaxation of the sphincter occurred only if the stimulus was very strong. Cocaine influenced only the dilator, and eserine only the sphincter. Because the latter drug did not cause an active relaxation of the dilator, but stretched the muscle, the pain so often observed following large doses of eserine can be accounted for. From these experiments the author concludes that the double innervation of the sphincter as postulated by Poos does not exist in the human eye.

P. C. KRONFELD.

THE SHARE OF EACH MUSCLE OF THE IRIS IN THE PUPILLARY REACTIONS. F. Poos, Arch. f. Ophth. **125:** 308, 1930.

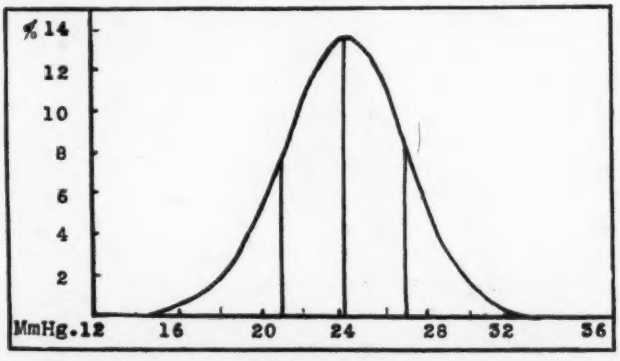
This is F. Poos' reply to the paper by C. Behr on the same subject. Poos contends that Behr's case was not suitable to demonstrate the relaxation of the sphincter under the influence of sympathomimetic drugs, which Poos has shown convincingly by experiments in vitro.

P. C. KRONFELD.

Physiology

THE NORMAL LIMITS OF INTRA-OCULAR PRESSURE MEASURED WITH THE SCHIÖTZ TONOMETER. H. K. MULLER, Arch. f. Augenh. **104:** 89 (Jan.) 1931.

The author has used the figures reported by Gjessing (*Arch. f. Ophth.* **105:** 221, 1905) on the intra-ocular pressures of over 2,000



eyes, and on the basis of these has constructed a curve of relative frequencies. The physiologic values range from 12 to 36 mm. of mercury, but a glance at the curve shows that 67 per cent lie between 21 and 27 mm.

F. H. ADLER.

THE MODE OF ACTION OF OCULOMOTOR STIMULATION: I. E. ENGELHART, Klin. Wchnschr. **10:** 26 (Jan. 3) 1931.

The practically accepted fact that a reaction to vagus stimulation is not due to the vagus alone but, in addition, to a substance produced by the stimulation, led to this investigation. It was hoped that such a substance might be found in the ocular fluids due to oculomotor stimulation. In the ocular fluids of rabbits no difference was found from those of cold-blooded animals. A rabbit's eye in which physostigmine had been used was compared with a normal eye. It was found that the vitreous of the eye in which physostigmine had been used had an effect on the heart of a cold-blooded animal as did an alcohol extract, while the normal vitreous caused no reaction. The experiment was carried further by exposing one eye to daylight and keeping the other in darkness. Since rabbits show no consensual light reaction it was thought that the production of a substance due to oculomotor stimulation would be well controlled. The eye kept in darkness showed only a slight amount of substance or none, while the eye exposed to light was rich in this substance. The author believes such a reaction is comparable to the vagus effect on the heart, and considers it a humoral mechanism. Other experiments on similar reactions are being undertaken.

L. L. MAYER.

THE MODE OF ACTION OF OCULOMOTOR STIMULATION: II. E. ENGELHART, Klin. Wchnschr. **10:** 215 (Jan. 31) 1931.

This is the second article on the subject of oculomotor stimulation. The first appeared in the January 26 number of the same journal. An investigation of the iris and ciliary body isolated from the remainder of the oculomotor apparatus revealed no evidence of a substance which might aid in oculomotor stimulation. When the oculomotor nerve was intact, stimulation by an electric current revealed evidence of this vagus-like substance. Further work on this problem is to be published (*Arch. f. d. ges. Physiol.*)

L. L. MAYER.

Retina and Optic Nerve

A NOTE ON GONIN'S OPERATION FOR DETACHED RETINA. J. H. FISHER, Brit. J. Ophth. **15:** 317 (June) 1931.

The author draws an analogy between exclusion of the pupil, a condition in which the aqueous is excluded from the anterior chamber and in which iris bombé results, and what occurs after the Gonin operation. When the Gonin operation succeeds, it does so because, if the site of the puncture has been determined with sufficient accuracy, the retina falls back into apposition with the choroid, as the subretinal fluid draws out, in such a way that the portion in which the hole exists corresponds with the site of the puncture; the choroid at this point, responding to the stimulus of the cautery, pours out an inflammatory exudate which organizes and firmly attaches the adjacent part of the retina and in doing so forms, as it were, an annular synechia for the retinal hole. Some retinal fluid no doubt remains at first, so that the retina is now bombé to the vitreous; this residue of subretinal fluid in a short time is

carried off by the lymphatic and vascular channels of the choroid until the whole of the detached portion of the retina is reapposed; the "ring synechia" prevents its being renewed by fluid from the vitreous; the normal bulk is restored to the vitreous, at the same time, by the fluid from the ciliary secreting mechanism, now that it can no longer run to waste by draining through the retinal hole into the subretinal spaces, as it interruptedly did before the operation was performed. It would appear to be necessary to determine as accurately as possible, before operating in each case, to what part of the choroid the retinal hole will correspond when the subretinal fluid flows out, and to puncture with the cautery at this point in the overlying sclerotic; the height of the detachment in the region of the hole is presumably a factor in determining the spot selected for the puncture; the rather free use of a large cautery increases the chance of success.

W. ZENTMAYER.

A CASE OF TYPICAL BILATERAL RETINITIS CIRCINATA. P. VITO, Arch. di ottal. **38**: 180, 1931.

The author's patient was a man, aged 73, in whom poor vision had been noted for three months. Vision was 3/60 in the right eye and 2/60 in the left. Both eyes showed typical rings of white exudate surrounding the macular region. A year later the white spots were somewhat more numerous, with a tendency to become confluent. This was more marked after another year when a number of punctate hemorrhages were also seen, while other small hemorrhages had been absorbed. Absolute central scotomas were present and incomplete ring scotomas. A general examination gave negative results aside from moderate generalized arteriosclerosis which affected also some of the smaller retinal vessels. A chemical analysis of the blood and tests of kidney function yielded normal results. The case is the sixth to be reported in Italy. The author discusses the previously reported cases and the theories of pathogenesis, and concludes that changes in the vessels present the only determinable etiologic factor. A bibliography and four drawings of the fundus are included.

S. R. GIFFORD.

ANGIOMA OF THE CEREBRUM IN LINDAU'S (HIPPEL'S) DISEASE. G. F. ROCHAT, Klin. Monatsbl. f. Augenh. **86**: 23 (Jan.) 1931.

In all cases of Lindau's angiomatosis of the central nervous system hitherto reported, the tumors were found in the cerebellum or in the upper portion of the spinal cord. The case reported here showed a large angioma of the cerebrum in addition to a large cyst, the size of a pigeon's egg, and two small, solid angiomas in the cerebellum. The patient, a man aged 31 at the time of his death in 1930, had been under observation at the neurologic clinic of the University of Groningen, Holland, since 1919. The chief symptoms were psychic disorders, attacks of unconsciousness, during which he bit his tongue, spasms of the arms and legs and incontinence of the bladder and rectum. Bilateral choked disks and right-sided hemianopia existed; amaurosis followed. No angiomas of the retina were found. The pathologic examination of the brain showed a condition identical with that seen in cases of angiomatosis of the retina. The tumors of the cerebrum

and cerebellum were of the same structure. Several members of the patient's family had Hippel's disease. One brother had angiomatosis of the retina with angiomas in the cerebellum and the spinal cord; another brother had angiomatosis of the retina and a cystic angioma of the cerebellum; one son of a sister had angiomatosis of both eyes. These cases were reported by Tresling (*Klin. Monatsbl. f. Augenh.* 1920, vol. 64) and Rochat (*Ibid.* **78:** 601, 1927). This case furnishes proof of an involvement of the cerebrum in Hippel's and Lindau's disease.

K. L. STOLL.

SUBRETINAL CYSTICERCUS. DINULESCU and NESTIANU, Cluj. med. **11:** 419, 1930.

The authors report a case of cysticercus of the right eye with vision reduced to perception of light. Tension was 25 mm. Ophthalmoscopically a relief-like formation completely covered the disk. It was sharply defined and whitish gray and occupied half of the nasal region of the visual field. Temporally it covered also the macula and extended almost as far as the ora serrata. The formation presented horizontal movements, and in the nasal region also the scolex and neck of the parasite were visible. The feces contained no ova of the parasite. The eosinophilia of the blood amounted to 15 per cent.

N. BLATT.

Trachoma

A TRACHOMATOUS FATHER CONTAMINATES HIS THREE CHILDREN THROUGH THE AGENCY OF A MORAX DIPLOBACILLUS CONJUNCTIVITIS. J. SEDAN, Rev. internat. du trachome **8:** 62 (April) 1931.

The mother of three children had been treated for trachoma since 1927. As is the custom, the children were examined at the incipiency of the mother's condition, and at various periods following. The mother was advised as to the contagiousness and kept a rigid toilet, so that there were no signs of the children being contaminated. The father was away from the family until January, 1930. On his return to them, and also to the clinic, he, too, was advised of the seriousness of the trachoma that affected him. In May, the father presented himself at the clinic with a diplobacillus conjunctivitis. This infection lasted about three weeks. In July the three children were examined, and all showed a beginning trachoma. At the same time the father had a fresh exacerbation of trachoma. The author calls attention to a similar case which he reported to the League Against Trachoma, in January, 1926. He agrees with the late William Nicati, who was wont to repeat that "trachoma in the secreting stage is dangerous to all and more apt to be disseminated."

L. L. MAYER.

THE ETIOLOGY OF TRACHOMA AND THE NEW DIAGNOSTIC, THERAPEUTIC AND PROPHYLACTIC CRITERIA DERIVED THEREFROM. C. ADDARIO, Ann. di ottal. e clin. ocul. **58:** 1074 (Dec.) 1930.

The importance of Noguchi's discovery of Bacillus granulosis is emphasized, and the case of human inoculation reported by the author

(*Rev. internat. de trachome,* 1930) is reviewed. A strain of the Noguchi organism received from New York was inoculated by subconjunctival injection into the upper fornix of a blind eye. After five days there was diffuse redness and a velvety appearance of both tarsal conjunctivae. Eight days after inoculation the right eye, which had not been inoculated, became inflamed, and three days later both eyes were the same, with beginning follicles in the folds. After four months definite follicles were present on the palpebral conjunctiva and a definite clinical diagnosis of trachoma could be made. The process extended, involving the semilunar fold and caruncle, and after ten months the condition was diagnosed as trachoma by several ophthalmologists, one of whom knew nothing of the inoculation. No mention is made of corneal involvement. The author believes that the case adds the evidence necessary to a definite proof of B. granulosis as the cause of trachoma.

He has observed that acute trachoma, especially trachoma in children, accompanied by active inflammation, often undergoes a spontaneous cure with very slight permanent changes, while chronic trachoma without active inflammation has no tendency to heal. He believes that the use of 2 per cent silver nitrate and 10 per cent solutions or ointments of copper, such as he employs, acts by stimulating a sluggish inflammatory process which hastens healing. In the attenuated form which he calls follicular trachoma, expression of the follicles greatly hastens the process. Acute infections superimposed on trachoma often hasten the healing process in the same way. When true trachomatous pannus is present and in case of keratitis, the use of a vaccine of B. granulosis has apparently been of definite service. The pannus due to trichiasis is treated by surgical correction of the trichiasis.

S. R. GIFFORD.

Tumors

A RAPIDLY GROWING MALIGNANT TUMOR OF THE LACRIMAL GLAND. G. H. PEARSON, Brit. J. Ophth. **15:** 314 (June) 1931.

A Chinese man, aged 21 years, had a swelling of the right upper lid, which was observed forty days previously to that of the preauricular gland, which had been present for twelve days. The whole upper lid was swollen and projected almost at right angles to the face. A soft tumor mass was felt below the upper margin of the orbit. The region of the parotid gland was also swollen. Complete removal being refused, a biopsy was made. The removed tissue was made up of epithelial cells. There was an abundance of cellular mitosis. There was also some infiltration by lymphocytes and polymorphonuclear cells. The growth was moderately vascular.

W. ZENTMAYER.

TUMORS OF THE LACRIMAL GLAND. F. COLLEY, Brit. J. Ophth. **15:** 365 (June) 1931.

Less than three hundred cases of tumor of the lacrimal gland have been reported. The author's patient was a woman, aged 37 years. There was no history of injury. At the age of 35, she had first noticed a

small lump under the outer part of the right eyebrow. The eyeball appeared to be dislocated "out of the orbit," being actually pushed over the inferior margin of the orbit medialward. The movements of the eye were restricted almost completely. Vision equaled perception of light. An encapsulated tumor was found. It extended for about 35 mm. toward the apex of the orbit. It was easily removed. The only attachment was to the conjunctiva of the upper fornix. A good cosmetic and functional result was obtained. Vision equaled 6/5 part. Histologically the growth was a spindle cell (fibro) sarcoma.

The author discusses the origin and nature of tumors of the lacrimal gland. It would appear that the great majority are made up of a mixture of tissues of mesodermal and ectodermal origin and therefore should be regarded as mixed tumors. In some cases, however, the stroma is so definitely predominant that the mixed elements may be overlooked and a mistake in diagnosis made. W. ZENTMAYER.

SARCOMA OF THE CHOROID. P. VANCEA. Arch. d'opht. **48**: 43 (Jan.) 1931.

Vancea reports the case of a young man with acquired syphilis of some years' duration who complained of loss of vision when first seen, and who was found to have bilateral optic neuritis with bilateral absolute scotomas. Treatment for syphilis resulted in improvement of the neuritis of the right eye, while the left became complicated by detachment of the retina, which was unrelieved by three scleral punctures. In this case the early central scotoma and a whitish disturbance about the macula were the only early symptoms of choroidal tumor. Although these are recognized as most important in the early symptomatology of sarcoma of the choroid, this diagnosis was difficult in view of the syphilitic infection. Secondary glaucoma developed two and one-half months after the onset of symptoms, and sarcoma was found on examination after enucleation. Orbital recurrences occurred four, eight and ten months after operation. The author is convinced that the neuritis in the right eye was a toxic neuritis due to the tumor in the left eye, and he believes that its occurrence deserves inclusion in the symptomatology of sarcoma of the choroid and that it may suggest the rapid evolution of the tumor. S. B. MARLOW.

EPIBULBAR NEVOCARCINOMA WITH EXTENSIVE INVASION OF CORNEA. CASTROVIEJO, Arch. de oftal. hispano-am. **31**: 257 (May) 1931.

After a detailed review of the literature, the author describes an epibulbar tumor in a woman, 62 years of age, which had started two years before at the external limbus and had gradually involved almost the whole cornea. The tumor was vascular, flattened, pinkish white and of a lardaceous aspect. The tumor was of epithelial and not of connective tissue origin; the pigmented cells were epithelial, not differing from those found in nevi. The growth originated in a nevic cell, with subsequent atypical lesions in the other epithelial elements of the cornea. He concludes with remarks on the prognosis and treatment of these tumors.

C. E. FINLAY.

Uvea

TWO CASES OF MILIARY TUBERCULOSIS OF THE IRIS CURED BY RADIOTHERAPY. DIACICOV, MUNTEANU and GAVRILESCU, Cluj. med. 11: 496, 1930.

In recent years the part radiotherapy plays in tuberculous involvements of the eye has become more and more prominent, individualization being, of course, necessary in any case in order to avoid injurious sequelae. Irradiation of the eyeball ought to be performed with an exact technic and technically unobjectionable apparatus. If the amperage and potential are previously exactly determined, if adequate filtration is employed, and if the quality and quantity of the rays are exactly measured, the irradiation of the iris can be carried out without risk. Amenable to radiotherapy are those cases of tuberculous iritis that do not improve under tuberculin, phototherapy and other local and general methods. The authors have submitted two cases to radiotherapy, using the following technic: recumbent position of the patient, lead rubber for the protection of the surroundings of the eye, Coolidge tube, 150 kilovolt vertex tension and 2 milliamperes, 1 mm. Al filter, focal skin distance 23 cm., 3/5 of the skin erythema dose, with an interval of one or two days between the exposures, and intervals of from three to four weeks between the irradiation courses. In the first case, in which also the Wassermann reaction was positive, the antisyphilitic treatment proved a failure; so did the local and the tuberculin treatment, as well as the phototherapy. The radiotherapy then instituted brought about a cure with good acuity of vision. The same favorable course was run by a second case treated by radiotherapy, in that improvement began after the third exposure and was followed by complete healing on further irradiations, with vision 5/5 in both eyes. On the basis of these two cases, the authors come to the conclusion that radiotherapy exerts a very active therapeutic influence on tuberculous conditions of the iris. In addition, also the hypotonic effect of radiotherapy is to be emphasized.

N. BLATT.

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(From Summaries by the Readers of the Papers)

(Concluded from page 482)

THE RÔLE OF HEREDITY IN OPHTHALMOLOGY (Concluded). DR. MARNIX VAN DUYSE, Ghent, Belgium.

Familial Disease of Maternal Inheritance.—Color blindness: This is transmitted as a recessive. Partial achromatopsia may be present for red (protanopes), the commonest form, for green (deuteranopes) or for violet (tritanopes), although no examples of this last form have been noted. The spectrum in these cases shows a gap.

Dyschromatopsia: This is the commonest form of color vision disturbance. The patients fail to distinguish one or more or all colors, except when brilliantly illuminated, and confuse them in a weak light or when the hues are not very bright. This form must not be mistaken for the one already noted, as there is merely weakening but no actual loss of color perception.

Congenital Dyschromatopsia: Daltonism is the form of this disease associated with partial achromatopsia for red (anerythropsia) which is confounded with green. As a matter of fact, these two colors are distinguished, i. e., they do not give the same color sensation, being differentiated by luminosity or shade values. So, often enough, Daltonism is discovered by accident, as the subject has learned which objects in nature are called green and which red. If one diminishes the illumination, this inherent difference of shade between red and green disappears and the subject has no clue to the actual color. The colors thus balanced for red-green blindness are known as confusion colors. The defect is found almost exclusively in males. Horner (1876) first showed that it is transmitted exactly like hemophilia, as he had described it in 1829, a tainted father passing on the disease to his grandsons through immune daughters. The author has already called attention to the fact that direct transmission from father to son can take place when an affected male mates with a female who is a latent carrier, and that, from then on, several successive generations may remain immune. If their offspring are limited, one may not happen to find male children in each generation. Wilson first showed the sex-linked inheritance (Morgan).

Retrobulbar Optic Neuritis (Leber's Disease): Hereditary degenerative diseases of the optic nerve, not accompanied by cerebral lesions, become manifest at quite different age periods in life: at birth, in childhood, more often in youth or in early adult life. This condition is transmitted as a sex-linked recessive. Leber (1871) observed fifteen cases in four families. The disease involves both eyes at the same time, in a number of persons of one and the same family, at times between the ages of 18 and 25, and at others between 41 and 49. At first, the patient complains of headache and dizziness, and soon notes loss of vision. After a period varying from a few days to from three to six months, vision is much reduced, and some subjects become completely blind. In patients of the same family the disease runs practically the same course, in time, a fact which deserves to be borne in mind as it has been noted in other hereditary conditions. The amblyopia is purely central, with macular scotoma and partial or complete loss of color perception, and may go on to amaurosis. The peripheral field is normal, or may occasionally show slight concentric contraction. The disk shows hyperemia with slight vascular changes, if any, possibly a little veiling of the outlines, and later the characteristic pallor of the temporal half or quadrant. Von Hippel noted, as against the hypothesis of an inflammatory origin, that such a process could not be transmitted by heredity. Fischer suspected an increase in the volume of the pituitary gland, but Lagrange claimed that this could not cause pressure atrophy of the maculopapillary bundle (? P. H. F.). X-rays have failed to show any enlargement of the sella or other cranial anomaly. Berger thought of anomalous development of the sphenoid. Its ossification is completed by the age of 20 to 25, and this would explain the appearance of the disease just at this time. Roentgenography shows a narrowing of the optic canal in the members of an affected family, and this explains the compression and atrophy of the nerve. If, said Terrien, the neuritis is the result of bony compression, why does one not always and at the very first find a concentric contraction of the field, and how does one explain an apparently selective action of the bony compression on the central bundle of the optic nerve? Lagrange noted delayed coagulation time and thought this may show a genetic relation between Leber's disease and hemophilia which, as noted, is transmitted similarly, as a sex-linked recessive. Endocrine disturbance may also play a part, as indicated by the frequent first appearance of this disease at puberty. Treacher-Collins thought that there was an abiotrophy (Gowers) or special hereditary predisposition of organs already completely developed to undergo a precocious degeneration. Certain tissues as well, e. g., the thymus, have a definite predetermined life-span, and in certain pathologic cases this span is shortened, notably in familial choroiditis (Doyne-Tay), familial symmetrical degeneration of the macula, amaurotic family idiocy, retinitis pigmentosa, heredity cataract and central degeneration of the cornea. In typical instances of sex-linked heredity the females are usually free, but if they are heterozygotes or carriers they transmit the disease to their sons. According to the theory, a normal father and a carrier mother should have sons affected in the ratio of 1 : 1. Sometimes the disease is transmitted from father to son, or according to the dominant mode. Females are not

always immune, Nettleship's tables showing 20 per cent women. Girls are not supposed to be affected unless the father is diseased and the mother a carrier. In Leber's disease the fathers of affected females are almost invariably normal. This may be explained by manifest heterozygotism or, according to Waardenburg and Kawakami, by changement of dominance, just like that noted in certain cases of hemophilia, daltonism, nystagmus, etc. Female carriers are numerous, and the sisters of affected females may transmit the disease to their sons, a fact that does not square with the theory that half the females are immune. If affected and of manifest heterozygotism, females can transmit the condition directly, and it can also go from father to son, although rarely, through from three to five generations. Early appearance suggests the factor of "anticipation" and constitutes a transition to the infantile form of congenital optic nerve atrophy. It is then difficult to say whether a particular case is congenital or a precocious degeneration of the optic nerve. This disease is related to the tapetoretinal degeneration of infancy in which optic atrophy is the first symptom. Congenital optic nerve atrophy might be considered as a degeneration coming on ante natum. These cases are rare, affect sisters and brothers and rarely carry through more than two or three generations.

Essential Heredofamilial Nystagmus: This is often a secondary manifestation, appearing together with hereditary anomalies of the eye (albinism, achromatopsia, lack of macula) or central nervous system (Friedreich's ataxia, nystagmus, epilepsy), but may appear as an idiopathic disease. In the latter case, vision may remain near normal, but, as a rule, it is weak. In certain families the nystagmus appears with amblyopia in some cases and with normal vision in others. The light sense and the appearance and color of the macula should be studied carefully with red-free light in every case of suspected congenital hereditary nystagmus.

Vogt's Makulalosigkeit (absence of the macula): This condition, noted in total ocular albinism or albinism of the fundus oculi alone, may easily be overlooked. Transmission is either as a dominant or as a sex-linked recessive (Nettleship). The first type is transmitted to children of the opposite sex, from father to daughter and from mother to son, and the affected children are decidedly less numerous (selective fecundation of Hemmes). Oscillations of the head are often noted as a complication, but these are not invariable, and as they have been met with in cases in which there was no nystagmus, it is not possible to establish a causal relationship between the two manifestations. In the recessive sex-linked type, transmission is typically and exclusively by females to male children.

Essential Familial Congenital Nystagmus (Myoclonia). This condition has been observed in families with as many as ten individuals affected. The sex influence is marked. Transmission is usually by immune females and males who are affected. The nystagmus sometimes appears after some febrile disease. In some forms there is an association with tremors of the lids, face and upper extremities. In one case, autopsy and histologic examinations failed to reveal any significant pathology.

Megalocornea: This was for a long time confounded with hydrophthalmos. Horner first showed a sharp difference between the two. Kestenbaum distinguished their features, as follows: In megalocornea there are no functional disturbances, the cornea is transparent and there are no breaks in Descemet's membrane. The disease affects men almost exclusively, and is almost always bilateral. The eyes are equally large, and the cornea has a normal curve. Embryotoxon is often seen. The malformation is, one might say, invariably familial, is transmitted as a sex-linked recessive and is never associated with hydrophthalmos. The latter is associated with increased intra-ocular tension, and later with excavation of the nerve head. Males and females are affected in the ratio of 5:3. The disease is monolateral in 35 per cent of the cases; the dimensions of the two eyes are not identical, and the cornea is flattened. Embryotoxon is never seen, the familial distribution is not so frequent, and the anomaly is transmitted as a simple, not sex-linked, recessive. Megalocornea is sharply congenital, and the anomaly shows no changes in the entire course of life of the subjects who, it is to be noted, retain good vision.

Ocular Diseases of Undetermined Hereditary Type.—1. Congenital Malformation: Anomalies of this sort, noted at birth, may in a way be contrasted with familial diseases which appear later and are progressive. (*a*) Coloboma: Failure of the optic slit to close at the normal term, at the end of the first or the beginning of the second month of fetal life, may be due to intra-uterine inflammation (Deutschmann), a theory difficult to reconcile with sharp localization and uniform symmetrical involvement of both eyes, to the presence of atypical mesoderm in this neighborhood (von Hippel) or, as generally admitted and supported by anatomic, histologic and clinical data, to atypical and rudimentary anlage of the ectoderm (von Szily). Cross-mating of colobomatous buck rabbits with normal does, and vice versa, proves hereditary transmission to a large number of the offspring by pathologic changes in the germ cells, which also determines various other malformations, such as atypical coloboma, microphthalmus or even complete anophthalmia, with the whole series of transitional forms, showing identity of the genotypic determinant. Goldschmidt thought that quantitative variations in the latter may explain variations in the degree or extent of the hereditary damage. Various forms of malformation may be noted in one and the same family; an identical anomaly is by no means always noted, but it is generally predetermined and prevalent. These anomalies are not examples of atavism, but according to von Szily, the expression of action of determinants in the proper sense, which, having been lost in the course of ontogenetic development, suddenly reappear (latent characteristics of the biologists). These characteristics cease to be recessive or become manifest by homozygotism, by a change of dominance, by the freeing of factors or by the disappearance of inhibiting factors. Possibly there is a kind of mutation that can be transmitted by heredity, as suggested by colobomas produced experimentally in animals by the action of poison, x-rays, etc. Atypical Conus: This is a true coloboma. Tertsch has demonstrated that inferior conus is associated with malformations of the retina, pigment epithelium and choroid. Microphthalmus: Besides the extremely

rare true form (ocular dwarfism), there is a form associated with and due to coloboma. Anophthalmia: The ocular anlage may be completely missing, but most of the time there is a rudiment between which and microphthalmus there is practically no difference. Formerly the condition was attributed to amniotic bands, but that theory is practically ruled out by the association with normally developed muscles, lids and orbit. Coloboma with an orbital cyst, microphthalmus and anophthalmia all derive from malformations of the ectodermic anlage. Colobomas of the Fundus: Typical choroidal coloboma is rare, appears related to congenital split formation in the iris, and is probably transmitted as a dominant. Coloboma of the optic nerve sheath has been produced experimentally by mating animals with coloboma of the iris, so that it probably has the same genotypic basis. Microphthalmus is transmitted as a dominant characteristic. It may be associated with myopia and corectopia. Coloboma of the Lens: This often is associated with ectopia lentis and coloboma of the iris and probably is due to the same genotypic determinant. Coloboma of the lens is transmitted as a dominant. A true hereditary malformation is always related to modification of the idioplasm. Still, Pagensteher and van der Hoeve produced true hereditary coloboma by experimental intoxication of the ovum. Von Szily, however, contended that these cases were not due to intoxication but to spontaneous, accidental mutations. Seefelder was of the opinion that an idiotypic malformation such as true coloboma may develop under the influence of a paratypic disturbance, as shown by the frequent coexistence of a typical coloboma with cyclopia, a paratypic malformation. If the two appear simultaneously, they must have a common origin, but one must be careful in applying to man the data obtained by animal experimentation.

(*b*) Malformations of the Iris: Persistent pupillary membrane and congenital split formation in the stroma of the iris are due to mesodermic anomalies of development. True polycoria is an ectodermal defect, extremely rare, and shows several pupils, each with a sphincter and a dilator. Aniridia is rarely complete. There is generally a stump adherent to the trabecular ligament, filling the angle of the anterior chamber and interfering with the off-flow of the aqueous. It is associated clinically, as well as genetically, with coloboma of the iris. Association of this anomaly, as well as of corectopia, dyscoria, lens coloboma and ectopia and congenital cataract, with aplasia of the macula shows an arrest of the development not only of the retina, but of all the ectodermal elements of the eye. (*c*) Malformations Due to Amniotic Anomalies: The factor of amniotic bands plays no part in the production of true ocular colobomas, but is limited in action to the fissures of the face, producing coloboma of the lids and lacrimal passages and, occasionally, deeper disturbances.

2. Errors of Refraction and Strabismus: Franceschetti insisted that to understand the rôle of heredity in these cases, one must reverse the usual order of investigation, and instead of starting with clinical and statistical data, begin with the general mendelian laws, see whether the facts noted in ophthalmic practice square with them and, if occasion arise, try to find out why they do not. The problem is, above all, one of general biology. Certain hereditary characteristics, e. g., height, can

be expressed numerically. If one measures a sufficient number of individuals of a heterogenous population, they can be arranged according to height in groups, each of which includes a certain number, and each of these corresponds to one of the coefficients obtained by developing the binomial $(1 + 1)^n$; distribution follows the binomial curve. In the eye, all parts capable of varying in size comport themselves in just this way; thus, the refraction of the cornea (Steiger) and other optical constants, such as thickness of the lens, anterior and posterior curve, anteroposterior axis of the globe and total refraction of the dioptric apparatus (Tron). The hereditary characteristics are influenced by the surroundings in which the living being develops (paravariation) and also by crossing (mixovariation). It has been possible to determine definitely the influence of paravariation by determining the refraction of genotypically identical (univitelline) twins. The differences noted hardly exceed 2 diopters, so more marked discrepancies must be in relation with mixovariation. The optical constants probably depend on several determinants, as is the case for other normal characteristics, such as size, coloration of skin and hair, etc. It is still undecided whether the optical constants depend on several determinants (polymerism) varying in quantity and acting en masse (Goldschmidt) or whether divers determinants, in part associated, enter into play. In the transmission of refraction, crossing does not result in a simple disjunction, and in the second generation the hybrids (F_2) do not constitute a middle term in relation to the progenitors (P); their characteristics at one time approach those of the father, at another, those of the mother. This has also been noted in regard to size and figure. If the parents present an exaggerated corneal curvature, this is also noted in the offspring, and one may infer that the same holds good for other optical constants. Refraction, being influenced by several determinants, cannot very well be transmitted according to a simple mode, say, as a dominant. We cannot compare the hereditary transmission of refraction with that of a single optical constant, as it is a state in whose make-up a number of determinants are concerned. This being so, does refraction in its distribution follow the binomial curve? It might well do so if the optical constants on which refraction depends were transmitted independently of each other and there were no correlation between them. Franceschetti took the following analogy to explain correlation and facilitate interpretation of the refraction distribution curve. He considered, arbitrarily, the length of the vertebral columns in relation, on the one hand, with the height, and on the other, with the breadth, of the individual vertebra, both of which dimensions vary according to the binomial curve. The length of the vertebral columns will vary similarly. If a correlation presents between high and narrow, or between low and wide, vertebrae, the curve of variation referring to the length of the vertebral columns will no longer correspond to the binomial curve. The correlation will give rise to the production of too large a number of columns of medium dimensions. If we imagine an absolute correlation, the columns will have an identical structure and dimensions, and there will be no statistical distribution. If the correlation between height and width of the vertebrae is negative, the curve will be modified, this condition favoring the production of columns of average dimen-

sions, and become very steep, i. e., high and narrow, while a positive correlation is favorable to bipartition and produces a low, broad galtonian curve. On the basis of 12,000 eyes, not counting high (degenerative) myopia, Scheerer and Betsch constructed a curve of refraction which was comparatively symmetrical but steeper than the binomial curve. The number of cases varying slightly from the normal (+ 0.5 diopters) is proportionately too large. Franceschetti interpreted this fact as a tendency of nature to reproduce emmetropia, the causal factor being a correlation between the simple factors on which refraction is based, distributed independently according to the binomial curve. The steep appearance corresponds to a negative correlation between two factors capable of increasing or diminishing the amount of refraction. There is a similar correlation (Tron) between the anteroposterior diameter of the globe and the total refraction power of the dioptric apparatus, the two factors varying inversely in a way to favor the production of emmetropia, refraction diminishing as the axis lengthens.

Myopia: In matters of heredity, this anomaly of refraction cannot be considered as a simple pathologic character, as it is only a partial manifestation. Donder's definition of myopia, based on physics and physiologic optics, is necessarily inexact and can be applied only to a relatively small number of cases, as biology alone can teach us what is to be understood by a normal characteristic. As such it considers a character which manifests itself in the majority of individuals, developing in the usual conditions of life. Refraction, like other hereditary characteristics in man and animals varies according to the binomial curve (Steiger). The latter, more marked at its summit, corresponds also to hypermetropia and low degrees of myopia, so that the latter must be considered as normal states. Most authors think that myopia depends on two factors, the length of the anteroposterior axis and the refraction of the cornea, particularly the first, which vary according to the binomial curve. If there were a marked correlation between the degree of myopia and the length of the anteroposterior axis, the latter should always be found to be too long in all myopic eyes. The existence of myopia without change in axis or with an axis shorter than normal is possible if the correlation is not marked, so that one cannot contend any longer that low myopia is always associated with elongation of the axis of the globe due to external causes. As to variations in total refraction, if there is a marked correlation between the latter and the myopia, the eye can be myopic only if the refraction is below normal; but if eyes with normal or supernormal refraction can be myopic, external causes cannot give rise to myopia by reducing the refraction of the cornea. There is a positive correlation of average importance between axis length and degree of low myopia (Tron) which explains why the length of the axis is generally greater in myopic than in hypermetropic eyes. If myopia could be reproduced by external causes, the relationship would be much more marked. Where hereditary characteristics are susceptible of quantitative variation, we frequently note in the offspring a return to parental characteristics. Hence, one would expect to see myopia appear in the descendants of a family in which the disease has been noted frequently, unless an opposite refraction state had attenuated its effect. Low myopia apparently acts as a dominant (Clausen

and Holm) in relation to emmetropia. Apparently, a character developing under the influence of several determinants does not generally behave like a simple characteristic and so, after a crossing, the hybrid character of the second generation will no longer be a middle term between the parental characters but will approximate that of one of the ancestors.

High Myopia: There seems to be two types. The first, not over 7 diopters, varies according to a symmetrical curve. The second, degenerative form, shows a more marked correlation between elongation of axis and degree of ametropia, although there are cases of myopia of upwards of 10 diopters in which the axis was only from 20 to 25 mm. long. The anomaly may be transmitted as a recessive. If this factor is simple, the children of persons with a high degree of myopia should all be myopic, which, in fact, seems to be the case (Clausen, Fleischer, Jablonski). As low myopia, on the other hand, is very widespread, the influence of consanguinity, so striking in high myopia, can only be minimal. The appearance of myopia in the course of several successive generations has suggested a dominant transmission, but, said Lenz, we must not forget that direct transmission of disease is not absolute proof of dominant transmission. It may be due to great frequency of the disease, and the number of latent carriers (conductors) may be considerable. Often, after two or three generations, a myope may marry an immune (female) carrier. The number of affected offspring will then be considerable, so that we may think we have to deal with a dominant. This becomes more probable if we are able to establish a correlation with other anomalies of admittedly dominant type, such as nystagmus, hesperanopia or poor sight. It may be sex-linked. Degenerative myopia often appears in association with ocular conditions belonging to the group of ectodermic malformations, such as albinism, ectopia of the lens and opaque nerve fibers, or with feeblemindedness (Best), and might also be considered as a hereditary degenerative disease. Errors of refraction are more common in idiots and epileptic dements than in normal subjects.

Hypermetropia: Low degrees are transmitted as a dominant. The parents of hypermetropic children are generally both hypermetropic, or at least one is (Krusius). High hypermetropia, like high myopia, is transmitted as a recessive and is to be considered as a pathologic condition, associated, as it often is, with an abnormally small cornea, cataract or other malformations. It should be classed with microphthalmic eyes, whereas high myopia is in a class by itself.

Concomitant Strabismus: Its frequency in the population is estimated at from 1 to 2.5 per cent. The children of normal parents are in the proportion of 1.2 per cent. The mode of transmission has not been definitely determined. It must be borne in mind that paralytic squint may be hereditary and by overaction of the antagonist bring on, secondarily, a concomitant squint. An anomaly of refraction with unilateral amblyopia may be a factor of practical importance. Crzellitzer found hypermetropia in over 86 per cent of his cases. It has been found impossible to separate, as to heredity, the factors of ametropia, squint and lack of development of fusion sense. The mode of transmission may be digenetic (Crzellitzer), polygenetic and, generally, recessive (Clausen and Baur), or dominant, as indicated by direct trans-

mission during several generations (Zande, von Sicherer). The multiplicity of factors explains why strabismus does not appear in the same form in all subjects. There may be favorable and unfavorable factors, so that one or more generations may be immune. Late transmission by apparently normal subjects relates to an irregular dominance. The relation of divergent strabismus to myopia is much less definite (60 per cent, Crzellitzer). Simple dominance is certainly rare, as indicated by the fact that only 47 per cent of the offspring are affected when one of the progenitors had squint, instead of 50 per cent, the normal proportion in dominance.

Familial Diseases of the Neuro-Ocular Apparatus: Heredofamilial Ophthalmoplegia: The term, created by Mauthner, denotes paralysis of at least two muscles innervated by two different nerves, or of two ocular motor nerves. By extension it has sometimes been applied to the paralysis of a single nerve when several muscles were involved, as in paralysis of the third nerve, whether total, involving both the extrinsic and the intra-ocular muscles, or partial and either external or internal. The site of the lesions is extremely variable: at the sphenoidal fissure, at the base of the skull or at the nuclei of origin.

Ophthalmoplegia Externa: This is congenital or acquired. Paralysis of the external muscles is generally congenital, but not always complete from the moment of birth. Frequently, very slight contractions of the affected muscles allowing a minimum of motion in certain directions can be detected. An error of refraction, nystagmoid contractions or true nystagmus usually accompany the acquired form of ophthalmoplegia. When muscles, normal at birth, show signs of beginning paralysis in youth, ptosis is generally the first sign, with gradual extension until the eye becomes completely incapable of motion. The hereditary paralyses, congenital and acquired, are transmitted according to the dominant mode, and direct transmission through several generations has been observed repeatedly. In the acquired form of ophthalmoplegia, coming on late, the homochronous nature of the heredity has been noted, but the biologic pathogenesis of these cases is very obscure. Total external ophthalmoplegia is much less frequent than isolated paralysis of a single muscle. Like total ophthalmoplegia, ptosis may be congenital or appear some time after birth (tardy ptosis of Dutil). In both cases the transmission is by dominance. Congenital ptosis, generally bilateral, is often accompanied by paralysis of other muscles, notably of the superior rectus, and is associated with total external ophthalmoplegia or with other pathologic manifestations, such as nystagmus or epicanthus. Here, too, the transmission is by dominance.

Tardy Ptosis of Dutil: This condition appears between the ages of 40 and 60, developing and progressing slowly and becoming complete only in the course of several years. The transmission is dominant (homochronous).

Hereditary Cerebellar Ataxia: This group is manifestly familial (Pierre Marie) and may be present clinically in various forms, either as cerebellar disturbance of the lower extremities (a staggering or dragging gait) or of the upper limbs (pseudotremor of the hands), soon followed by more evident disturbance in the motions of the head and

trunk. Optic atrophy, often bilateral, is one of the commonest associated symptoms, and another, less usual sign is static ptosis, present only when at rest and disappearing with movement. Transmission is by the recessive mode, and the subjects, normal at birth, become affected toward the age of 40 in the first generation, and at an even earlier age in succeeding generations (anticipation). (The author noted that a complete bibliography on heredity in ophthalmology was given by Clausen [*Zentralbl. f. d. ges. Ophth.* **13**: 1731, 1924] and by Franceschetti [Kurzes Handbuch für Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1].)

HEREDITY IN A FAMILY WITH RETINITIS PIGMENTOSA. DR. GASALLA, Lugo, Spain.

The transmission was at first recessive, and later sex-linked (matriarchial). One female of this family was twice married, both times to normal males, and had, by both, sons who were affected with retinitis pigmentosa. In this family there were no consanguineous marriages nor was there a single case of retinitis pigmentosa in the female. The affected males, seven in number, noted the first symptom, hesperanopia, at widely varying ages, one not until 35. The last generation is, so far, immune, but the disease may not be extinct, and may yet appear.

WHAT IS THE VALUE OF AN EYE? DR. H. VILLARD, Montpellier.

The author noted that certain jurists have opposed the observations of ophthalmologists and have tried to show that the accidental loss of one eye has no effect on work or on earning capacity. His conclusions on this important medicolegal subject are: 1. The loss of one eye does alter visual function. 2. This alteration lessens professional capacity. 3. The figure of this diminution, in the first few years following the loss of the eye, should be fixed at from 25 to 33 per cent, depending on whether the occupation followed was nonvisual or visual. 4. Lessening of earning capacity due to loss of one eye improves to a certain degree with time. The exact extent of improvement could be determined only by a follow-up of the injured workmen and by investigation of their earning capacity and final income. This could best be done by a world-wide questionnaire, in collaboration with ophthalmologists, which it would be well to entrust to the International Labor Bureau.

RELAPSING PURULENT IRIDOCYCLITIS. DR. L. CARRÈRE, Montpellier.

A man, aged 27, without significant personal or family history, had had for more than a year attacks of iridociliary inflammation, now in one eye and then in the other; these were sluggish, cleared up without sequelae and reappeared about every month. Tuberculosis, syphilis, gonorrhea and rheumatism can be excluded. The author inclines to the theory of intestinal toxemia or hemosporia; temporary cure, at least, having been effected by diet, intestinal disinfection and oral administration of vaccines.

SEVERE SYMPATHETIC OPHTHALMIA: TEMPORARY IMPROVEMENT STARTED BY AN ATTACK OF THE GRIP. DR. SEXE, Besançon.

A chip of steel from a hammer penetrated the left eye. In spite of early extraction of the foreign body (eight days after injury), sympathetic ophthalmia developed in the other eye. An emergency enucleation of the left eye was performed. For thirty-eight days intensive treatment was kept up, but without avail, with intravenous injections of cyanide (of mercury? P.H.F.), a proprietary arsenical product and salicylate of soda, thirty-eight subcutaneous injections of pilocarpine nitrate and fifteen deep orbital injections in the region of the optic foramen of the left eye. A severe attack of influenza on the thirty-ninth day stopped all treatment. Spontaneous and progressive improvement occurred for eleven days. Then, an attack of secondary glaucoma took place in the sympathizing right eye. An iridectomy caused cessation of pain but noticeable loss of the sight that had been gained. At the end of treatment, the patient was hardly able to see his way about. The author called attention to similar partial cures by intercurrent disease, and cited the case of a recruit with hereditary syphilis recommended for discharge on account of bilateral keratitis with complete pannus. He had an attack of intercurrent measles, and, after six weeks, there was clearing of the corneas with vision 8/10. The author also called attention to the effect of a fresh attack of erysipelas on varicose ulcers, and to the malaria treatment for dementia paralytica. Further observation of this sort might be of value. His own attempts to cure heredospecific interstitial keratitis by vaccination failed in all twelve cases. The inoculation invariably failed to "take."

DIAGNOSIS AND PROGNOSIS OF MELANOTIC TUMORS OF THE IRIS. DR. V. MORAX, Paris.

At the beginning it is not always possible to distinguish, by a single examination, a pigment patch from an actual new growth. It is the progress and development of the colored spot that will decide the question, and for the determination and comparison of the dimensions of the lesion at various times, photography of the anterior segment of the globe is essential and indispensable. The development of melanotic sarcoma is often extremely slow, as we know from many case reports. As vision may remain unaffected for a long time, there is no need to enucleate in a hurry, especially as melanotic tumors in the region of the iris, in contradistinction to those of the choroid, appear to give rise much less frequently to viscereal metastasis.

THE ZONULE OF THE VERTEBRATES. DR. M. TEULIÈRES AND DR. J. BEAUVIEUX, Bordeau.

Histologic microscopic sections and study with the slit-lamp show that the zonule is a simple suspensory ligament keeping the lens in place in those animals in which the fibers of the zonule are inserted only at the equator or in its immediate vicinity. In these cases, there is little or no accommodation. In monkeys and man, on the contrary, besides this supporting rôle, the wide insertion of the zonule, extending to some distance on both surfaces of the lens and not limited to the

equatorial ridge, indicates its importance in the function of accommodation. This may also apply to day birds of prey, whose zonular insertions are particularly well developed.

CATARACT OPERATION WITH VERTICAL SECTION. DR. JACQUEAU, Lyons.

The author used in fourteen cases—and as a result, highly recommended—the procedure of Junes of Sfax, Tunisia. After the usual preparation with a slight retrobulbar anesthetic injection, the author performs (1) a puncture of the cornea with the lance knife in the region of the limbus on the temporal side, sufficient to allow the easy introduction of the blunt end of a pair of small scissors curved on the flat side; (2) a scissors section, upward and downward, in one step, of the flap at the limbus; (3) a wide cystotomy, and (4) expression of the lens. The advantages of this method are the complete docility of the patient, who does not have to be exhorted repeatedly to look down. There is no pulling on the globe, and the field of operation is reached with the greatest ease. The section heals very rapidly, the corneal flap fitting itself at once into the scleral margin and preventing prolapse of the iris. There is great ease of access again if for any reason it should be necessary, during the first few days, to add one or two touches. The astigmatism is vertical, and the patients become used to it more easily than to the horizontal form. There are no serious objections. Good pupillary dilatation is important, as is a good clean section. At first, there may be a little trouble in expressing the lens because of our being used to making pressure exclusively from below, whereas now it must come from the nasal side alone.

NEW METHOD OF INTRACAPSULAR EXTRACTION OF CATARACT. DR. I. ABRAMOWICZ, Vilna, Poland.

The flat spoon of Smith is passed behind the lens and carried down over one third of the posterior surface, and the cataract extracted by means of a modified form of Weber's loop, laid flat on the anterior surface of the lens.

IMMUNITY AND EXPERIMENTAL INOCULATION CATARACT. DR. LAVAGNA, Nice.

Inoculation of guinea-pigs with an emulsion of human cataract, when intra-ocular or near the eye, produced cataract in 72 per cent; when subcutaneous or buccal, in 55 per cent. Inoculation of a gram-positive diplococcus, a facultative aerobic organism isolated from forty human cataracts, produced 80 per cent and 56 per cent, respectively, of positive results, i. e., cataracts, under similar conditions. After immunization with a microbic vaccine prepared from isolated layers of the human lens, the receptivity of guinea-pigs for experimental cataract was reduced to 45 per cent and 15 per cent, respectively. A specific virus may be the pathogenic agent in cataract. At all events, these experiments seem to show that senile human cataract, quite like the experimental inoculation cataract which resembles it strangely, is particularly amenable to treatment with a specific antigen.

ECTOPIA LENTIS AND GENERAL MALFORMATIONS. DR. G. WEILL, Strasbourg.

In eight cases this developmental anomaly was associated with coloboma of the iris or lens, calcification of the lens and arachnodactylia of the giant or dwarf variety. These anomalies are not due merely to disturbance in the development of mesodermic tissues but to a dystrophy of all the embryonic tissues and particularly affecting the endocrine glands, on a basis of ancestral syphilis, consanguinity, alcoholism or intoxication.

SENSITIVITY OF THE LENS TO X-RAYS. DR. NORDMANN, Strasbourg.

In contradistinction to the view, held up to ten years ago, that the lens was relatively immune to the action of roentgen rays, numerous positive results in the form of experimental cataract in adult animals have proved that the lens is, on the contrary, the most susceptible part of the eye. Clinically, cataract is seen rather often after roentgen treatment, and Jess has published cases in which it developed after irradiation of the temples and neck for deep-seated tumor when the rays had had to traverse the bones of the skull. The author's case was that of a young boy who had been treated for microsporia by a single application of x-rays for epilation and who presented bilateral cataract of characteristic roentgen type. There had also been an extensive x-ray dermatitis.

INDUSTRIAL INJURIES DUE TO FOREIGN BODIES. DR. BADOT, Jumet.

Accurate localization is of great practical importance in cases of foreign body. Wessely's shells and the stereoscopic triad greatly aid roentgenography. Copper and iron are badly borne by the intra-ocular structures, chalkosis and siderosis being the special pathologic states induced. Glass, on the other hand, causes very little reaction.

RELAPSING VITREOUS HEMORRHAGES IN A YOUNG ADULT. DR. HAMBRESIN, Brussels, Belgium.

Bilateral recurrent hemorrhages occurred in a young man of 28, in connection with tuberculosis. Besides the usual treatment, the common carotid was ligated and x-rays applied on one side. As a result there has been only one more hemorrhage in the eye on that side, while in the other eye there were ten or more. The author called attention to the frequency of bacillary diseases of the eye in apparently healthy subjects and not in admittedly tuberculous patients. Ligation of the common carotid and irradiation of the globe, and possibly also of the spleen, appear to be of great value in the treatment for these hemorrhages of the vitreous.

CHANGES IN THE RETINAL REFLEXES. DR. J. L. PAVIA, Buenos Aires, Argentina.

The author laid stress on slight changes in the reflections from the limitans interna as one of the earliest signs of choroidal lesions which otherwise show practically nothing. This is borne out by stereoscopy

with the Nordenson-Zeiss retinagraph, which gives pictures of the fundus with or without retinal reflexes according to the incidence of the illuminating rays. In a case reported, the choroidal disease was indicated by irregularity of the perimacular reflexes which became normal when the patient was put on a diet for excess blood nitrogen. Some time later, the patient having neglected to keep to his diet, the irregularity of the reflex was noted again with a greater loss of vision. At present, under the renewal of a strict dietary regimen, the reflexes have become regular, and vision and field have improved. In the second case, a woman had a disease of both optic nerves, probably due to overlooked syphilis. In the right eye there was a peculiar appearance of the upper and inner portion of the perimacular reflex which was broken up by small linear, radiating reflexes. There was hypercholesterolemia, slight lowering of vision, concentric contraction of the field for white and colors, and slight hemeralopia. After treatment, this all cleared up, and the radiating reflexes could hardly be found.

SURGICAL TREATMENT OF DETACHMENT. DR. JEANDELIZE AND DR. BEAUDOT, Nancy.

Statistics of operative results with Gonin's method within two years were given. Tear and disinsertion were found in 75.5 per cent of the cases; operable cases amounted to 59.1 per cent. A cure (retina reattached) was obtained, with vision from 1/10 to 4/10, in 20.8 per cent; improvement, with vision from 1/10 to 4/10, in 45.8 per cent; the total percentage of good results was 66.6 per cent. Relapse, i. e., a new tear at a point distinct from the first, coming on after a period of cure of from three to ten months, occurred in 25 per cent. Recurrences cloud the remote results of the method, which, however, is the best that we have at present. We lack, above all things, a form of medical treatment which would act on the choroid and retina and on the vitreous. Mercurial medication is of great value, even in cases in which there is no question of syphilis.

A CASE OF DETACHMENT SUCCESSFULLY TREATED BY GONIN'S METHOD. DR. PAUL PESME, Bordeaux.

Spontaneous detachment occurred in the myopic eye (right) of a woman of 57. Situated above and inward, the detached zone presented at its center a single large tear. Thermopuncture (Gonin) was performed three days after the onset of the accidents. The immediate result was excellent, with complete reattachment. The thermopuncture scar, alone, persisted in the form of an oval white patch which included in cicatricial tissue the original retinal tear. Four months after operation, vision was 4/10. The galvanocautery causes less traumatism of tissue and gives as good an adherent cicatrix as the thermocautery. Besides its action in obliterating the tear, the author thinks it is of value in reducing by cicatrical contraction the excess curve of the sclera, thus making it easier for the retina to reapply itself in contact with the wall of the globe. Time will tell whether this feature is really to be put to the credit of the method, but the highly encouraging immediate result should by all means be noted.

CASES OF DETACHMENT: THE GONIN OPERATION. DR. P. VEILL AND DR. M. A. DOLLFUSS, Paris.

This is a report of eleven cases. Cauterization of the tear was done with a special galvanocautery or, preferably, with the thermocautery. In seventeen cauterizations, of which several were on the same eye, there were no unpleasant complications, and all the patients were up by the tenth day, unless another operation was performed. In eleven cases of comparatively recent (from three months to eight days) detachment, there were four complete cures of several months' standing, two markedly improved cases and one failure. Four other patients who are improved will have to be operated on again to close the tear, as this is the sine qua non for definite cure.

THE TREATMENT OF DETACHMENT. DR. ARRUGA, Barcelona, Spain.

This is a study of the subretinal fluid that was removed in two cases of recent detachment and that had the exact appearance of fluid vitreous. The author presents a detailed study of tears during their formation. A minute lance-shaped instrument is introduced into the interior of the eye and controlled by ophthalmoscopic examination which notes its position relative to the tear. This reading determines the site of application of the cautery with much more accuracy. Etiologically, the author lays stress on the origin of detachment in a retinal degenerative or inflammatory lesion with a tendency to tissue retraction. The tear in the retina put the vitreous in contact with the choroid, which has the faculty, which the retina has not, of absorbing fluids, and, accordingly, the liquid part of the vitreous. Furthermore, movements of the globe successively widen the tear. Cauterization acts mainly by sealing off the choroid from the vitreous.

THERMOPUNCTURE IN DETACHMENT COMPLICATED BY ABSOLUTE GLAUCOMA. DR. M. A. TERSON, Paris.

A patient had intense pain in an eye which long before had shown a detachment and, later, extremely high intra-ocular tension. A conjunctival flap was raised, and a postequatorial ignipuncture of the sclera practiced. The pain was relieved, and the eye has remained quiet for about two years. Many cases of increased tension in detachment yield to dionin and miotics or, if not, to sclerotomy, iridectomy and other anterior operations. Still we may well keep to sclerochoroidal thermoperforation for cases of absolute glaucoma, primary or secondary, hemorrhagic or otherwise, and thus avoid removal of the eye.

FUNCTIONAL EXAMINATION OF THE CENTRAL RETINAL ARTERY. DR. DUBAR, Paris.

The author and Dr. Lamache showed the great importance of the factor of spasm in the abnormal resistance which the artery offers to compression when tested with the dynamometer. This measure, easily applied clinically, gives valuable information as to the condition of the cerebral circulation.

ARTERIAL RIGIDITY AND THE PHYSIOLOGY AND PATHOLOGY OF THE RETINAL CIRCULATION. DR. FRITZ, Mons.

By simultaneous clinical measurement of the rigidity, caliber and blood pressure of the central retinal artery we can distinguish four circulatory types: 1. Most cerebral hemorrhages and those into the vitreous occur with a low degree of rigidity, high pressure and normal or slightly increased caliber. The fundus is normal and vision good except for the hemorrhagic lesions. Vasodilators are very effective; vasoconstrictors, dangerous. 2. Persistent lowering of vision and optic atrophy are met with in high degrees of rigidity, low pressure, normal caliber or slight irregular contraction. Vasoconstrictors are indicated, but are not very effective; vasodilators are contraindicated. 3. A passing visual defect is noted in cases of slight rigidity and low pressure. Vasoconstrictors are very effectual. Dilators are dangerous, even for the condition of the general circulation. 4. Compensated hypertensions, without visual defect, are associated with high rigidity and normal or slightly reduced caliber. In extreme cases this condition leads to hypertensive retinitis with hemorrhages at the points of least vascular resistance and arterial obliteration. Vasodilators are indicated, but are not very effectual.

RETINAL ARTERIAL TENSION IN NASAL DISEASE AND IN PERIPHERAL (BELL'S) FACIAL PARALYSIS. DR. G. WORMS AND DR. G. CHAMS, Paris.

After endonasal operations we note regularly a disturbance of retinal arterial pressure ending in a phase of hypotension, and this, too, in the absence of any previous ocular lesion. This sheds light on the vasomotor factors in the cure for retrobulbar neuritis of accessory sinus or other origin. Some lesions of the nasal fossae relating mainly to the septum and exciting irritation reactions, such as coryza, paroxysmal sneezing and rhinorrhea, are often associated with retinal hypertension with or without ocular disturbances, contraction of the field, central or sector-like scotoma, arterial spasm, partial or complete optic nerve atrophy or the syndrome of retrobulbar neuritis. Operative removal of the irritating nasal spur is followed, almost invariably, by noticeable improvement and, at times, by complete cure of the ocular complications. Atrophic rhinitis, on the other hand, is always almost associated with retinal hypotension which remains homolateral when the nasal process is limited to one side and does not give rise to any ocular trouble. This would indicate a sympathetic factor in the pathogenesis of atrophic rhinitis.

THE VESTIBULORETINAL REFLEX. DR. G. WORMS AND DR. G. CHAMS, Paris.

In Bárány's test by irrigation of the ear, retinal arterial hypertension is induced on the same side (twenty-five seconds), rapidly followed by a fall and a longer phase of low pressure (forty-five seconds) with a return to normal. Subjects with recent labyrinthine irritation show retinal hypertension of the corresponding side. In these

cases, Bárány's test provokes a retinal arterial hypertension, of very slight degree, followed by a more marked hypotension than under normal conditions. With recently lowered excitability of the labyrinth we find retinal arterial hypotension, which is not affected by Bárány's procedure. In old labyrinthine lesions retinal tension does not seem, as a rule, to be affected.

PHOTOGRAPHY OF THE FUNDUS: ITS ANATOMIC, PHYSIOLOGIC AND CLINICAL IMPORTANCE. DR. J. MAWAS, Paris.

The author uses the Zeiss-Nordenson apparatus, based on Gullstrand's principle of centralized ophthalmoscopy, and he lays stress on the geometric method which led to various improvements (Bagneris, Dimmer, Thorner) in construction, notably the avoidance of reflexes, the separation of the pupil into two distinct halves, one for the illuminating rays, the other for the emergent rays to the photographic sensitive plate. The method is of great value in the clinical record of retinal changes and their variations, the accurate measurement of the caliber of vessels, and the follow-up of the clinical progress of disease.

EXPERIMENTAL STUDY OF THE ACTION OF α-METHYLACETYLCHOLINE ON THE IRIS AND INTRA-OCULAR TENSION. DR. M. VILLARET, DR. J. BESANÇON AND DR. J. GALLOIS, Paris.

The authors reported results of animal experimentation (reserving clinical data for later publication) confirming Reid Hunt's observation of a miotic action. This is also noted when the drug is combined with bromocholine, histamine, yohimbin, ergotamine or pilocarpine. Atropine neutralizes the miotic action of the choline derivative, but the latter abolishes the mydriasis caused by ephedrine. There is, at least in animals, a marked diminution in intra-ocular tonus. Details were given as to the subconjunctival and subcutaneous administration of α-methylacetylcholine.

SPONTANEOUS FLUCTUATIONS IN NORMAL INTRA-OCULAR TENSION. DR. MARC AMSLER, Lausanne, Switzerland.

A systematic study using clinical tonometry, twice daily, showed fluctuations averaging 8 mm. of mercury with extreme figures of 3 mm. (stable tonus) and 16 mm. (unstable tonus). A single application of the tonometer, no matter how exact, does not give a true picture; only a tonometric curve, based on repeated tests, will give a fairly accurate conception of the régime of intra-ocular tension. There is no one curve, normal and typical, which would serve as a theoretical starting point for studies having in view the experimental modification of intra-ocular tension. According to individual conditions, intra-ocular tension may be stable or unstable.

THE CARDIOVASCULAR SYSTEM IN GLAUCOMA. DR. J. ROLLET, Lyons.

A systematic and minute study of the heart and peripheral vessels in glaucomatous subjects showed lesions in almost every case. Moderate hypertension, especially, was very frequent, suggesting the importance of vascular factors in glaucoma.

NOTES ON THE MEDICAL TREATMENT OF GLAUCOMA. DR. C. LACAT, Paris.

The author obtained excellent results with Abadie's combined method (ergotin, epinephrine, calcium chloride) with pilocarpine drops; it relieved symptoms, prevented progress of the disease and improved vision in cases in which pilocarpine, used alone, was disappointing. Five patients treated in this way and followed up for three years gave him complete satisfaction, and he was able, without danger, to stop treatment for several weeks, after it had been used regularly for a number of months. In simple (essential) chronic glaucoma, this would seem to be the treatment of choice, but it is unsatisfactory in secondary glaucoma. The author reported the case of a man of 44. At 18 he had been completely cured of a serious attack of interstitial keratitis by means of a long course of injections of cyanide of mercury. At the age of 44 symptoms of glaucoma presented. Pilocarpine and the combined treatment of Abadie proved ineffectual, and, considering the syphilitic factor in the past history, the patient was put on almost continuous intravenous injections of mercury cyanide for six months. The symptoms disappeared like magic, and vision was 2/3 in each eye.

ACUTE BILATERAL RETROBULBAR NEURITIS DURING INTENSIVE ACETARSONE (STOVARSOL) TREATMENT. DR. M. NIDA, Paris.

A patient, aged 42, had an old case of syphilis with painful pseudo-anginoid crises and roentgen evidence of aortitis and dilatation of the left ventricle. Intravenous injections of mercury cyanide did not give the hoped-for results, and neoarsphenamine intensified the crises. Subcutaneous injections of sodium acetarsone were given, two of 1 Gm. and then regularly 1.5 Gm., twice every two days, to a total of 15 Gm. The first series was well borne, and resulted in a complete disappearance of the cardiac symptoms, but the second course, also totaling 15 Gm., started two months later and finished in the comparatively short space of twenty days, was followed by an attack of bilateral retrobulbar neuritis with vision 1/4. This cleared up of its own accord with a return to normal in six weeks (?). Nida warned of the danger of exceeding the dose limit of sodium acetarsone previously indicated by Sezary and Barbe in the treatment for secondary syphilis, viz., 1 Gm. subcutaneously, three times a week for seven weeks (?).

PAPILLORETINITIS: MASSIVE VITREOUS HEMORRHAGE IN SYMPATHETIC RETINITIS TEN YEARS AFTER INJURY OF THE EXCITING EYE. DR. FRADKINE, Autun.

The author thought that some marked disturbance of general condition is needed to explain the preparation of the field for this dramatic explosion at so late a date. Marked arterial hypertension had induced tension (intra-ocular? P.H.F.) in the stump of the injured eye and acted as a predisposing cause of sympathetic ophthalmia in the other. The opticochiasmal path seems to be the usual one for the transmission of sympathetic irritation and inflammation, which, in this case, was limited to the posterior half of the globe, and was definitely cured by enucleation of the exciting eye, with autohemotherapy.

PSEUDOTUMORAL (EDEMATOUS) PAPILLITIS IN ARTERIAL HYPERTENSION. DR. P. ABRAMI, DR. JEAN GALLOIS AND DR. FOUQUET, Paris.

Several cases were reported in which the disk changes indicated were suggestive of tumor of the brain or chronic nephritis, but the diagnosis was undecided in the absence of histologic examination. Certain authentic cerebral tumors can occasion marked arterial hypertension, and, conversely, arterial hypertension may of itself give rise to a syndrome of intracranial tension with papilledema and erroneously lead to surgical intervention. In the absence of focal, localizing symptoms, no one manifestation is pathognomonic, whether it be arterial hypertension, intracranial pressure or albumino-cytologic dissociation. The existence of marked retinal hypertension in a case of papilledema might point toward a nephritis. In a doubtful case ventriculography might throw light on the diagnosis.

FIVE CASES OF TUMOR OF THE HYPOPHYSIS. DR. E. TAHINDJIS, Constantinople.

Five patients had serious lesions of the optic nerves, changes in the sella and hemianopia. Four were operated on by Hirsch of Vienna according to his method, with encouraging results. On the other hand, the fifth patient, who was treated by deep roentgenotherapy, after successive periods of apparent improvement, more or less prolonged, had relapses. Finally, roentgenotherapy ceased to have any effect on the intracranial tumor.

THE HYPERTHERMIA-PALLOR SYNDROME IN OPHTHALMIC SURGERY IN NURSLINGS. DR. MOREAU, St. Étienne.

This reaction, a dangerous possibility, may come on in the forty-eight hours following an operation on a nursing infant. Few cases are on record following an ocular operation. A baby of 2 years, operated on for infantile glaucoma, developed, six hours later, temperature of 39 C. (102.2 F.) and then of 40 C. (104 F.), and died twenty-five hours after operation in a condition of pallor. This syndrome is much more common in otorhinology, as operations in that field are so much more often performed on children in the first years of life. With Dr. Beutter, the author reported (Congress of Pediatrics, 1929) ten cases in which four patients recovered. Histologic examination of two suprarenal capsules showed acute necrosis of the medullary layer. Without operative traumatism the syndrome of hyperthermia-pallor may occur spontaneously in a well child. Several reports deal with eczematous nurslings. Preventive measures, preoperative and postoperative, previously suggested, are of little value. Beginning six hours after operation, the temperature is taken hourly, and according to the indications of the curve, cold spongings and baths are given with injections of camphor, ether or guaiacol in oil. If the temperature continues to rise, one or more lumbar punctures are done. The etiology and pathogenesis of this syndrome are entirely unknown. It has a certain medicolegal interest.

PLEXIFORM NEUROMA OF THE ORBIT AND UPPER LID. DR. MARIN AMAT, Madrid, Spain.

An infant of 28 months had presented since birth a tumor formation involving the left orbit and upper lid with marked proptosis and good vision. The clinical signs were those of hard, separate movable nodules, not adherent to the skin and giving the impression of being made up of bundles of twisted and intertwined fibers. Extirpation of the neoplasm in its two portions, orbital and palpebral, was complete in one piece, showing that the growth had begun as a sort of pedicle at the apex of the orbit and was made up of the lacrimal, nasal and frontal nerves, entirely separate at the origin but fused together, a short distance from it, by an intertwining of its branches to form the new growth. On section the appearance was not unlike that of half-cooked macaroni. Histologic examination by Dr. del Rio Hortega, showed threads of nerve tissue surrounded by connective tissue, with preservation of a few medullated nerve fibers undergoing degeneration.

ULTRAVIOLET RAYS IN OPHTHALMOLOGY. DR. M. MOUTINHO, Lisbon, Portugal.

Under Wood light the normal lens shows fluorescence and a bluish-white color like cataract. The illumination of the pupil in case of dislocation of the lens, or very fine membranous aftercataracts might literally throw (ultraviolet) light on the presence or absence of structures in this region, and be of diagnostic and operative importance. As an aid in extraction of a lens dislocated into the vitreous, this illumination is very useful, the more so as fluorescence appears, whether we use natural or artificial light. After extraction of soft cataract this light shows the presence or absence of lens masses in the anterior chamber. The method should become a routine in operative procedures involving the lens.

Directory of Ophthalmologic Societies *

FOREIGN

British Medical Association, Section on Ophthalmology

President: Mr. E. E. Maddox, Glenartney, Poole Road, Bournemouth, England.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 7-9, 1932.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: July 17-21, 1932.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Leipzig. Time: June, 1932.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: New Orleans. Time: 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. F. Barnhill, Miami Beach, Fla.
President-Elect: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.
Place: New London, Conn. Time: June, 1932.

SECTIONAL

New England Ophthalmological Society

President: Dr. S. Judd Beach, 704 Congress St., Portland, Me.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Walter F. Hoffmann, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. Claude T. Uren, City National Bank Bldg., Omaha.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Fontanelle Hotel, Omaha. Time: November, 1931.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. C. Ward Ellis, 215 N. Walnut St., Lansing.
Secretary: Dr. H. B. Weinburgh, 301 Seymour St., Lansing.
Time: Third Thursday of alternate months.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the Month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. M. H. Merriman, 115 Prospect St., Waterbury.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: W. L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. W. O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas F. Wood, 74 S. 9th St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. L. Wicks, Valley City.
Secretary-Treasurer: W. L. Diven, National Bank Bldg., Bismarck.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193, 11th St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. W. R. Thompson, 600 W. 10th St., Fort Worth.
Secretary: Dr. A. F. Clark, 414 Navarro St., San Antonio.
Place: San Antonio. Time: December, 1931.

Utah Ophthalmological Society

President: Dr. Franklin H. Raley, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. L. Smith, 1005 Medical Arts Bldg., Salt Lake City.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. C. P. Jones, Newport News.
Secretary-Treasurer. Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. G. A. Smith, Montgomery.
Secretary: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Charles W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Earl LeRoy Wood, 192 Roseville Ave., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society. Section on Ophthalmology

Chairman: Dr. Alan C. Woods, 906 Cathedral St., Baltimore.
Secretary: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. John N. Evans, 23 Schermerhorn St., Brooklyn.
Secretary: Dr. Michael J. Buonaguro, 589 Lorimer St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. A. F. Luhr, 40 W. North Ave., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Frank E. Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. E. Burner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadephia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Ivor Clark, 188 E. State St., Columbus, Ohio.
Secretary-Treasurer: Francis W. Thomas, 327 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. F. M. Sulzman, 1831, 5th Ave., Troy.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Forth Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robison, Eye, Ear, Nose and Throat Hospital, Houston.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. B. J. Larkin, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. R. R. Montgomery, 102 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Russell T. Uhls, 823 Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Herbert F. Wolters, 130 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. William P. Haney, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. A. Cassiday, 1620 Medical Arts Bldg., Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Martin H. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Scott C. Applewhite, 705 E. Houston St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. W. F. Swett, 490 Post St., San Francisco.
Secretary: Dr. Robert C. Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. Claude L. La Rue, Highland Clinic, Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. M. G. Brown, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse.
Place: University Club. Time: Second Friday of each month except June, July and August.